IAN W. FRENCH & ASSOCIATES LIMITED

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HEALTH IMPLICATIONS
OF
EXPOSURE OF UNDERGROUND
MINE WORKERS TO DIESEL
EXHAUST EMISSIONS

Prepared for
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by

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HEALTH IMPLICATIONS OF EXPOSURE OF UNDERGROUND MINE WORKERS TO DIESEL EXHAUST EMISSIONS

1.0 PREFACE

This report was prepared by Ian W. French and Associates Limited under contracts (1982) #14SQ.23440-1-9003, (1983) #23SQ.23440-9-9143 and 03SQ.23440-2-9062 for the Department of Energy, Mines & Resources, Canada Centre for Mineral and Energy Technology (CANMET).

The document is an integration and expansion of an earlier review of the world literature, published December 11, 1978, also titled "Health Implications of Exposure to Underground Mine Workers To Diesel Exhaust Emissions" (1). The present review was completed in essentially the same manner as the 1978 edition, with the inclusion of published information which has become available since completion of the original document. More reliance has been placed on meeting abstracts for current information, particularly for research which is ongoing but not yet complete and fully reported in the open literature.

In the present up-date, the section headings of the original document have been retained, but new headings have been added.

The primary purpose of this document is to critically review the relevant scientific literature describing the occupational environment to which underground miners are exposed and to evaluate the potential for this environment to adversely affect the mine workers' pulmonary function and health. In particular, emphasis is placed on the potential or real health effects of gaseous and particulate emissions resulting from the use of diesel-powered equipment underground.

A brief review of the mechanical and chemical aspects of diesel engines, as well as their impact on the underground mine environment, as taken from the original presentation, are presented in this report for orientation purposes. The main thrust of the document is, however, related to the potential health effects to workers employed in "dieselized" mines.

CANMET requested that the report be prepared in such a manner as to permit it to be understood by those with scientific and technical backgrounds, as well as by individuals less familiar with the relevant medical or technical areas. Thus, introductory and explanatory notes and discussions have been included throughout the report. For example, general background information on topics such as diesel engines, pulmonary function, radiation, mutagenesis, carcinogenesis and dose-effect relationships have been included and will hopefully permit the unfamiliar to more readily understand the technical data and accompanying discussion necessary to such a technical review. Many of these sections were taken from the original document (1), with or without modification and the addition of new data.

As was stated in the 1978 preface, the most important and difficult issue to scientifically express is the potential additive or synergistic effects of the various constituents of the complex environment to which the miner is exposed. However, an attempt will be made to estimate the miner's health risk resulting from his exposure to the incredibly complex environment in the dieselized mine.

In the present document, the authors have attempted to further rationalize the development and application of the Ventilation or Air Quality Index introduced in the 1978 report.

C. Anne Mildon, B.A., M.D., D.O.H.S.
Ian W. French, B.S.P., Ph.D.

2.0 INTRODUCTION

Canada has almost one quarter of the total world and about 45% of the North American underground diesel population. This suggests that the Canadian mining industry should provide an excellent milieu for the epidemiological study of the health effects of diesel exhaust, particularly in metal and uranium mines.

2.1 Purpose Of Study

The Canadian Centre for Mineral and Energy Technology (CANMET), a group within Energy, Mines and Resources Canada, is engaged in research to assure the health and safety of Canadian mine workers by developing and making available technical knowledge necessary to meet or better existing standards. CANMET recognizes that health hazards may be present in the mining environment, and that differences exist between authorities as to the relative importance of the various contaminants found in the "dieselized" mining atmosphere.

Many reports on environmental conditions in mines have been published and health hazards associated with uranium and other mining activities were partially reviewed in the report of the Royal Commission On the Health and Safety of Workers in Mines (Ontario) (commonly referred to as the Ham Report). CANMET is, nevertheless, cognizant of an additional considerable medical literature related to the harmful and synergistic effects of the various mine atmospheric constituents such as diesel exhaust emissions, dust and cigarette smoking.

In order to review and consolidate the vast scientific and medical literature available in learned journals and books, CANMET commissioned Ian W. French & Associates Limited to review the relevant literature pertaining to the occupational environment in underground mines and the potential effect of the environmental components on the respiratory system of the workers. In particular, the work statement for the contract required a review of the progression of pulmonary function deterioration through to the development of frank respiratory disease that could occur as a result of the long-term exposure of miners to their underground environment. In addition, the authors were requested to critically review the medical evidence which might assist in the delineation of the absolute or relative importance of cigarette smoking and exposure to diesel exhaust fumes on pulmonary function and disability.

Realizing that there are many areas which are devoid of information or for which insufficient reliable scientific data is available to permit irrefutable conclusions to be drawn as to the effect of particular mine atmospheric contaminants on pulmonary function, CANMET required that the principal centres of research or knowledge in Canada and the U.S.A., and to a lesser extent in other parts of the world, be identified and areas which require additional study and investigation be indicated.

The concern of U.S. health authorities for the possible adverse health effects of diesel emissions deserves comment. John F. Finklea, M.D., Director of the National Institute for Occupational Safety and Health (NIOSH) communicated with Mr. Robert Barrett, the Administrator of the then Mining Enforcement and Safety Administration (MESA), in a letter dated June 7, 1976, in which a concern was indicated on the part of NIOSH for the possible health implications of exposure to diesel exhausts. In this letter, NIOSH stated that they "cannot offer any assurance that long-term exposure to coal dust and diesel exhaust at levels maintained within existing standards will protect the coal miner from adverse health effects." In the same letter, Finklea stated that NIOSH felt it "prudent to inform all concerned in the coal mining industry that further introduction of diesel equipment underground in coal mines pending completion of studies (on the health status of underground miners who have been exposed to diesel exhaust over a period of years) might result in future economic disruption should their use pose an unacceptable health risk." In response to this correspondence, MESA informed the coal mine operators in July, 1976 of NIOSH's attitude towards underground diesel equipment. The Director of NIOSH did, however, note that "we do not now have adequate human or animal studies to justify a recommendation at this time that existing diesel usage in underground coal mines be prohibited."

In appears that the cautious attitude displayed by NIOSH resulted, at least partially, from pressures placed upon them by the mining industry unions, who had clearly and publicly expressed their concern over the increased use of diesels in underground mines during a conference held in Pittsburgh in 1973 and published in the U.S. Bureau of Mine Report R8666.

At the same time, the U.S. Environmental Protection Agency (EPA) and the U.S. Department of Energy (DOE) and private corporations became concerned with the potential deleterious health effects which might result from the increasing use of light-duty diesel engines and thus funded research into the toxicity of diesel exhaust as it related to the ambient urban environment.

In view of these concerns of NIOSH, MESA, EPA and the DOE in the United States, CANMET's decision to carry out the present literature review was timely.

As was noted in the Preface, in December, 1978, Ian W. French and Associates Limited published the original document entitled "Health Implications of Exposure of Underground Mine Workers to Diesel Exhaust Emissions" (1) under a contract from Energy, Mines and Resources, which was a summary of the relevant world literature available to mid-1978.

In the conclusions to that review, several recommendations were put forward, including a ventilation formula which considered the major health hazards in dieselized mines. It was suggested that the formula might be employed to calculate a "ventilation" factor "V", which could provide an estimate of the adequacy of emission controls in the mine in providing an acceptable work environment.

In the past several months this formula has found application by various investigators (2) and government groups (3, 451), under the title of the "Health Effects Index" (HEI). More recently it has been recommended that the term Air Quality Index, or AQI, be adopted.

The "V" or "AQI" formula incorporated the ACGIH additive TLV concept used for mixtures of toxic compounds and provided recognition of the synergism between two gaseous components of diesel exhaust, SO_2 and NO_2, and the respirable particulate matter, or soot, present in diesel exhaust.

The formula was as indicated below:

$$\frac{(\mathrm{CO})}{\mathrm{TLV\text{-}CO}} + \frac{(\mathrm{NO})}{\mathrm{TLV\text{-}NO}} + \frac{(\mathrm{RCD})}{\mathrm{TLV\text{-}RCD}} + 1.5\left[\frac{(\mathrm{SO_2})}{\mathrm{TLV\text{-}SO_2}} + \frac{(\mathrm{RCD})}{\mathrm{TLV\text{-}RCD}}\right]$$

$$+ 1.2\left[\frac{(\mathrm{NO_2})}{\mathrm{TLV\text{-}NO_2}} + \frac{(\mathrm{RCD})}{\mathrm{TLV\text{-}RCD}}\right] = \text{"V" or "AQI"}$$

Since the publication of the 1978 review, a considerable volume of research has been started and/or completed into the chemical and toxicological characterization of diesel exhaust. Because of the current trend to the increased use of diesel cars and light-duty trucks, referred to above, much of the work relates to emissions from light-duty diesel engines. Data derived from these studies is applicable to the heavy-duty engines used in LHD mining equipment if the diesel components are similar and are present in equivalent concentrations in the exhaust of both types of engines or if the toxicological research has been carried out at component concentrations relative to those measured in the dieselized mine environment.

In view of this ongoing research activity, Energy, Mines & Resources (CANMET) contracted with Ian W. French and Associates to up-date the 1978 report and to modify the "V" or "AQI" formula, if necessary, to take into account new knowledge.

In addition to reviewing the published literature relevant to the toxicology and health effects of diesel exhaust, a thorough review of the presentations of the EPA meeting held in Cincinnati in December, 1979, was completed. Attendance at a meeting in Michigan in March 1981, sponsored by The Coordinating Research Council and publication of the Proceedings of the EPA 1981 Diesel Emissions Symposium held in Raleigh, North Carolina in October, 1981 (4), provided an up-to-date contact with investigators and legislators and provided new information on the chemistry and toxicity of diesel exhaust. Moreover, just prior to the completion of this document one of the authors (Ian W. French) visited with several of the primary U.S. diesel toxicology research organizations to ascertain the current status of their diesel-related research activities and to ascertain whether any additional, but as yet unpublished, data of relevance in estimating health risks to diesel exhaust was available.

The present document provides a summary of the relevant data and papers reviewed and makes further recommendations relative to the safe use of diesel equipment in underground mines.

Several investigators (especially at the Cincinatti and Raleigh meetings) addressed the problems of exhaust particulate and chemical composition, as well as methods for the characterization of diesel components. It is important when considering exhaust sampling that gaseous hydrocarbons tend to condense onto particulate surfaces during dilution and particulate agglomeration and hydrocarbon adsorption and desorption also occur upon cooling and dilution. Moreover, particulate size distribution varies with exhaust dilution so that studies carried out in dilution tunnels have to be carefully assessed as to their correlation with the practical world of exhaust dilution in a mine shaft. It is one thing to determine the chemical and toxicological properties of diesel exhaust components collected directly from the tailpipe or after dilution in an experimental tunnel, and possibly quite another to do the sampling within the mine atmosphere. A considerable literature has developed in the past few years defining the exhaust composition of smaller vehicles used for personal transportation and light-duty trucks. Because this equipment is operated under quite different conditions, particularly in relation to the fuel:air ratio, the data developed in terms of exhaust composition are not the same as for diesels used underground and will not be discussed in this report. However, where toxicological data has been developed using exhaust from these types of vehicles and where the quantitative and qualitative composition of the exhaust can be related to that known to be emitted by underground diesel equipment, these data are considered relevant.

Studies which explore the interaction of photochemical reactions with pollutants above-ground are also not applicable to the underground mine situation.

2.2 Notes on The Preparation of the Report

In order to satisfy the work requirements of the contract, Ian W. French & Associates began their literature review by carrying out several computer searches using various data bases such as Toxline, Medlars, Pollution Search, etc., and several key words pertinent to the environmental situation in dieselized mines. From this base, scientific papers were retrieved from the literature and reviewed for their relevance to the topics to be discussed. From the bibliographies in the published papers so retrieved, and from other documents available to the authors through Canadian, U.S., and other foreign government services, additional references to relevant scientific literature were retrieved and reviewed.

In order to familiarize themselves with the mining environment, which hopefully would permit a more pragmatic approach to the contract requirements, the authors visited two mines: International Nickel Company (INCO) metal mines in Sudbury, Ontario and the Denison Uranium Mine in Elliot Lake, Ontario. In addition to those indicated above, the authors also attended key meetings related to the health effects of diesel exhaust, including one in Chicago, Illinois in April, 1977 concerned with the nature and composition of diesel exhaust emissions and a workshop in September, 1977 in Morgantown, West Virginia on the health implications of the use of diesel equipment in underground coal mines. This latter meeting was sponsored by NIOSH and consisted of a gathering of eminent scientists from several countries and included medical and scientific personnel from a broad spectrum of disciplines. (This meeting is referred to throughout this report as the 1977-NIOSH Diesel Workshop or Meeting.) As well, one of the authors (CAM) was present at the 1979 EPA meeting held in Cincinnati.

Although the contract requirements did not include an investigatory or fact-finding commission, the authors nevertheless attempted to make the review as comprehensive as possible, within the financial and time limitations imposed, by seeking the opinion of various authorities. To this end, provincial and federal mining personnel in Canada, the U.S.A. and many countries around the world were contacted and requested to contribute any pertinent information and personal opinions related to diesel exhaust and the health of the underground miner. Those communications considered by the authors to be significant are included in this report, and appropriate references made to the contributor and his affiliation.

The work contract required that the authors "extract the practical knowledge in the scientific literature rather than review it in detail." In order to minimize the possibility that pertinent data might be omitted, an extensive search of the literature was carried out.

More than 4,000 scientific papers, reports and communications were indexed during the preparation of this document.

Of the approximately 3,250 papers reviewed, some were considered to be of minor or peripheral interest and are not abstracted or discussed in this report.

2.3 Canadian and World Use of Diesel Engines Underground

In papers presented at the 1977 - NIOSH Diesel Workshop, Stewart & Co-workers (5, 6), reviewed the history and present use of diesel-powered machinery in Canadian underground mines.

Dieselized equipment has been in use in Canadian coal mines for almost thirty years and their presence in mineral mines remains extensive. Zorychta (7) listed the first diesel-powered equipment used in a Canadian mine as being a 100 brake-horsepower (bhp) locomotive introduced underground in 1949. This compares to an estimated 8,675 - 10,000 units worldwide in 1976 (5, 8). More recently, one authority estimated there were about 22,000 vehicles in use in the world's underground mines and tunnels. Of the over 3,200 pieces of equipment in use in Canada, about 40% are used to power load-haul-dump (LDH) vehicles with rated horsepowers from 50 to over 300. An additional 20 - 25% are used for production haulage vehicles. The remaining 35 - 40% of the diesel engines are employed with light duty vehicles, such as drill jumbos, jeeps and service vehicles (5, 6).

Although the data avialble from Stewart et al (5, 6) do not permit an accurate calculation of the horsepower allottment to each category of vehicle, some 1977 figures from British Columbia and Ontario are of interest. In British Columbia, out of a total of almost 20,000 bhp underground, 43% was used to power LHD equipment. An additional 26% was used on front-end loaders and haulage vehicles, with the remaining 31% being driving service and personnel carriers.

The Ontario data, which was provided by International Nickel Company (INCO) (6), represented diesel use in their Sudbury operations. As of early 1978, INCO had 571 units in use underground with a total rating of 57,000 bhp. This included 255 LHD machines, which comprised 62% of the total diesel bhp in INCO mines. Locomotives for track haulage used 119 diesel engines, with a total bhp of approximately 5,950 or 10% of the total. The remaining 27% (or 15,575 bhp) underground were used in 197 other vehicles, including drill carriers (77 vehicles), haulage trucks (17 vehicles), and service and personnel vehicles (93 vehicles).

The INCO communication noted that most of the diesel units were powered by Deutz air-cooled indirect injection (IDI) engines with precombustion, swirl chambers.

Denison Mines Limited provided the authors with an inventory of the dieselized equipment used in their uranium mine operation in Elliot Lake as of January, 1983. Analysis of their data provided the following information. The total diesel bhp underground was 24,257, with the use being distributed as shown in Table 1.

TABLE 1

Distribution of Diesel Equipment at Denison Mines, Elliot Lake, Ontario (1983)

	Total bhp	Percent of Total bhp
Service Vehicles:		
123 units with 35 - 181 bhp, including lift trucks, jeeps, tractors, carriers, graders, etc.	10,695	29.0
Scooptrams:		
59 units with 180 - 242 bhp	11,712	31.8
Bolting and Drilling Rigs:		
85 units with 65 - 139 bhp	7,036	19.1
Ore Haulage Equipment:		
31 units with 210 - 270 bhp	7,410	20.1

(Source: Personal communication - Denison Mines Limited)

Of the 298 diesel engines in use at that time at Denison Mines, 183 or 61% were manufactured by the Deutz Engine Company. This represented 45.6% of the total diesel horsepower in use at the operation.

A discussion of the relative use of Deutz engines is relevant when considering the type of diesel engines contributing emissions to the Canadian underground mining atmosphere. On the basis of the data provided by INCO and Denison Mines, and from discussions with CANMET personnel, as well as Provincial mine inspectors it is clear that the bulk of diesel-related emissions in Canadian mines are from Deutz IDI engines or engines of a similar type.

Of the diesel-powered equipment used underground, the heavy-duty, high-horsepower LHD and haulage vehicles operate the greatest percentage of the time. Other dieselized equipment, such as drilling and bolting rigs, service and personnel carriers operate intermittently and have, on average, smaller engines and therefore contribute less to the total atmospheric content of diesel-related contaminants than the LHD-haulage vehicles.

Because of these considerations, the present report will stress the characteristics of IDI engines, and in particular Deutz engines. This contrasts with a NIOSH document (9), which was used as a foundation for the 1977 - NIOSH Diesel Workshop, in which a detailed discussion of diesel exhaust emissions was included for a wide variety of engines, including the IDI type, but no specific mention was made of the Deutz engine. Similarly, a report prepared for the American Mining Congress on the health effects of diesel exhaust (10) had relatively little specific information on the Deutz diesel, although it did discuss, in general terms, the IDI engine.

Dieselized equipment has been used in coal mines in Great Britain for many years and would seem to provide a suitable location for the study of the effects of diesel emissions. In a paper presented to the 1977 - NIOSH Diesel Workshop by J. D. Kibble of the National Coal Board (11), it was pointed out that there were 617 diesel locomotives in use in British coal mines. In addition, some 80 diesel tractors and supply vehicles were also in service.

As of July 1977, there were 162 diesel units in 28 U.S. coal mines, of which about 95% were located in the West Mississippi area. In non-coal mines there were about 4,000 diesel units in operation in 400 mines, which represented a use of diesel power by approximately 50% of the mine operators (12).

Clark, of the Australian Bureau of Mines, indicated that in his country a total of 599 diesel were in use underground as of 1976. These were mainly haulage vehicles and were used in "well-ventilated routes". Clark emphasized that the use of diesels at the mine face was limited and did not begin until 1966 (12).

In South Africa, where some 390,000 people are employed underground (13), there were about 3,000 diesel engines in use in 1977, most being haulage vehicles operated in through-airways. Vehicles of the LHD type are not used in gold and platinum mines, but are used in other mines, such as copper and coal. Diesel locomotives are, however, used in diamond, gold and platinum mining. A summary of diesel locomotives used in South African gold mines is given in Table 2.

TABLE 2

Diesel Locomotive Use in South African Gold Mines

Rating (KW)	Number of Units
5 - 15	64
16 - 30	1,739
31 - 45	1,011
46 - 60	277
60 - 75	5

(Source: Reference 13)

Detailed statistics of diesel use in other countries were not available, but Alcock (8) reported the following figures in late 1976: South America, 255; Western Europe, 2,100; U.S.S.R. and Communist Countries, 400; and Asia, 125.

For those who may not be familiar with diesel-powered mining equipment, Figures 1, 2 and 3 depict typical equipment. Figure 1 shows the bucket end of an LHD vehicle, with Figure 2 indicating the LHD-operator's position. Figure 3 is a photograph of a jumbo drilling rig, which is mobilized using a diesel engine but operated using hydraulic power.

FIGURE 1

LHD Vehicle - Bucket Loading

FIGURE 2

LHD Vehicle - Operator's Position

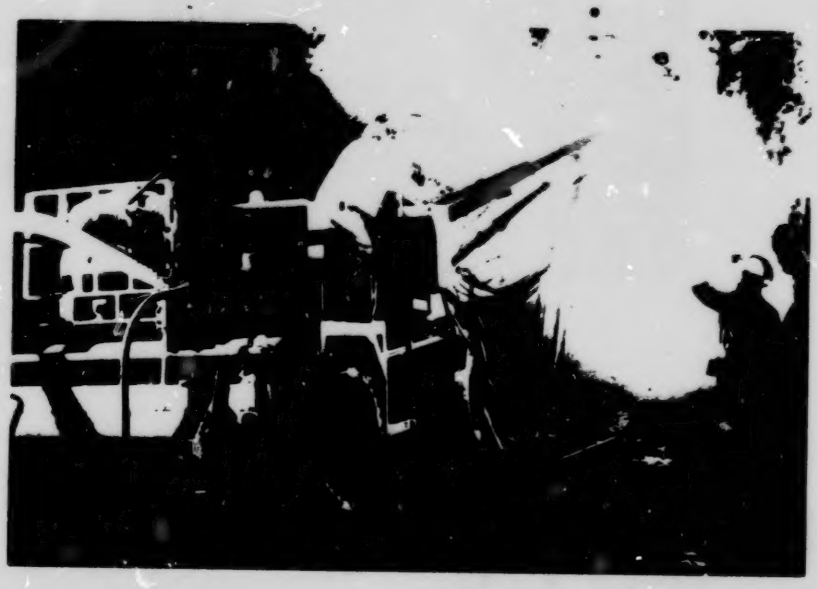


FIGURE 3
Drilling Rig

(Photographs courtesy of Denison Mines Limited)

3.0 INTRODUCTORY TECHNICAL DISCUSSIONS

As indicated in the introduction, the contents of this section are included to assist those readers who are less familiar with the technical, scientific and medical background required to fully understand the health implications of exposure to diesel emissions. Readers may choose to omit reading all or portions of these sections, depending on their experience and knowledge of specific areas discussed.

3.1 Pulmonary Structure and Function

The respiratory system of the human (Figure 4) starts simply and becomes amazingly complex. The system commences with the nose, which consists of two simple orifices, through which 500 ml of ambient air is inhaled with each breath, equating to about 10,000 l/day or 3,300 l per average 8 hour work period. Immediately the nasal passages expand with soft moist mucosa covering the passageways, plus small hairs in the mucosa. In this expanded passages are rolls and mounds of cartilage called turbinates which are covered with moist mucosa and sticky secretions.

The inhaled air travels with considerable turbulence down the main airway called the trachea, which is also lined with sticky mucous.

Inside the chest cage the trachea divides into two main bronchi, the right and left bronchus. Each main bronchus enters its respective hemithorax where the bronchus divides into successively smaller bronchi and bronchioles. This division at 23 different levels results in 630,000 small airways, called terminal respiratory bronchioles, leading into alveolar ducts and alveolar sacs called alveoli.

The main bronchi are 1.1 - 1.9 cm in diameter where inhaled air rushes through the airway with great turbulence which allows impaction of large particles on the airway walls. When the air passes into smaller airways, the turbulence diminishes and when the air reaches the small airways of less than 2 mm in diameter, the air flow is smooth and quiet and designated as laminar flow.

Finally, these airways end in the distended pouches of respiratory membranes called alveoli (Figure 5), where air currents have slowed greatly and small particles move with rapid rates of Brownian movement so that

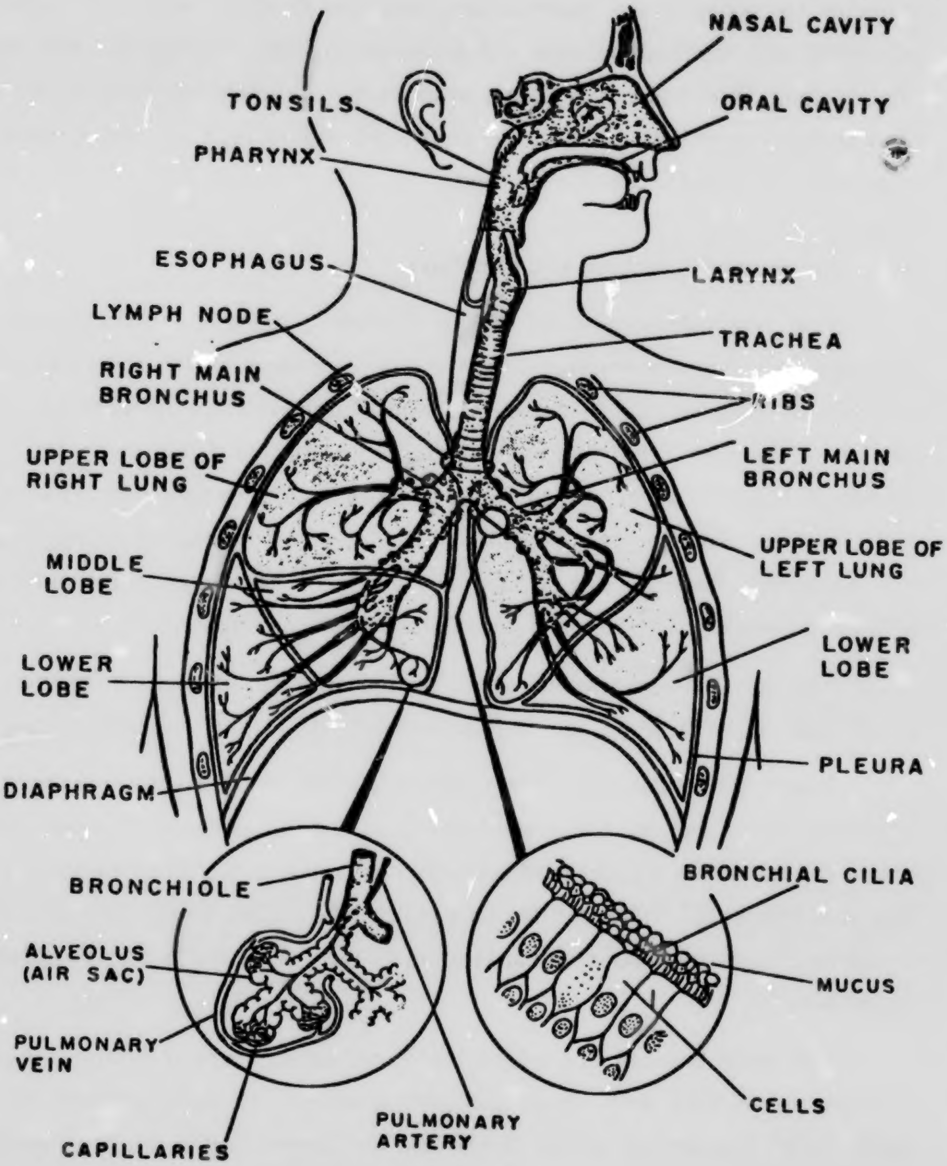

NASAL CAVITY
TONSILS
ORAL CAVITY
PHARYNX
ESOPHAGUS
LARYNX
LYMPH NODE
TRACHEA
RIGHT MAIN BRONCHUS
RIBS
UPPER LOBE OF RIGHT LUNG
LEFT MAIN BRONCHUS
MIDDLE LOBE
UPPER LOBE OF LEFT LUNG
LOWER LOBE
LOWER LOBE
PLEURA
DIAPHRAGM
BRONCHIOLE
BRONCHIAL CILIA
ALVEOLUS (AIR SAC)
MUCUS
PULMONARY VEIN
CELLS
CAPILLARIES
PULMONARY ARTERY

FIGURE 4

The Respiratory System

(Source: Reference 16)

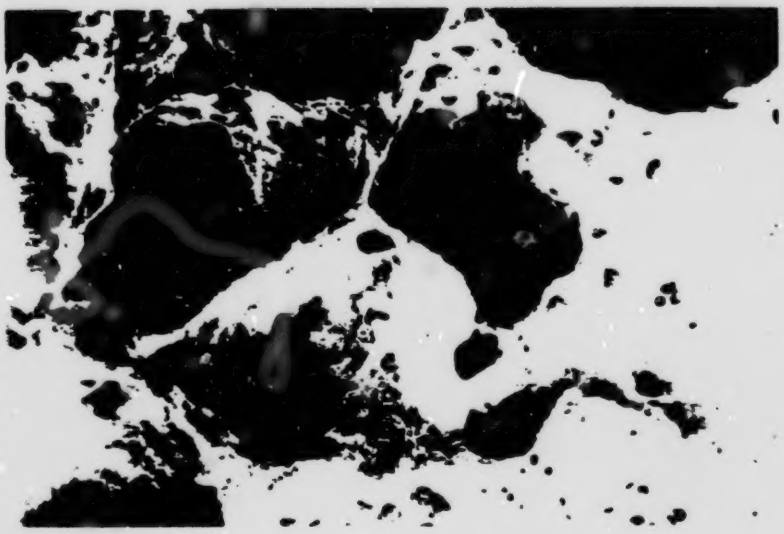


FIGURE 5

Scanning microscopic photograph of alveoli
(From American Lung Association Bulletin, April, 1974)

deposition is by diffusion. Respiration takes place in the alveoli where oxygen is exchanged for carbon dioxide according to the body's needs (15). The relationship of the alveoli with the blood is diagrammatically and microscopically presented in Figure 6. Figure 7 provides a schematic depiction of oxygen passage from the alveoli to the blood.

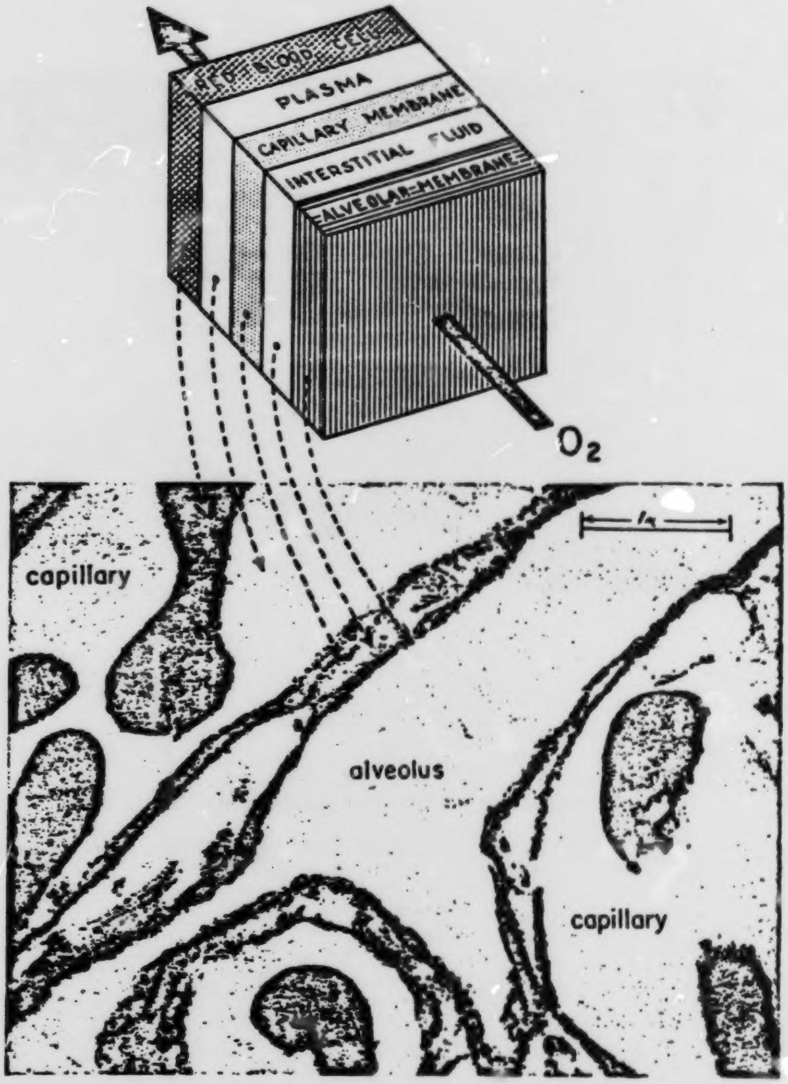

RED BLOOD CELL
PLASMA
CAPILLARY MEMBRANE
INTERSTITIAL FLUID
ALVEOLAR-MEMBRANE
O_2
1μ
capillary
alveolus
capillary

FIGURE 6

Electron Microscopy of Rat Lung

(Source: Reference 17)

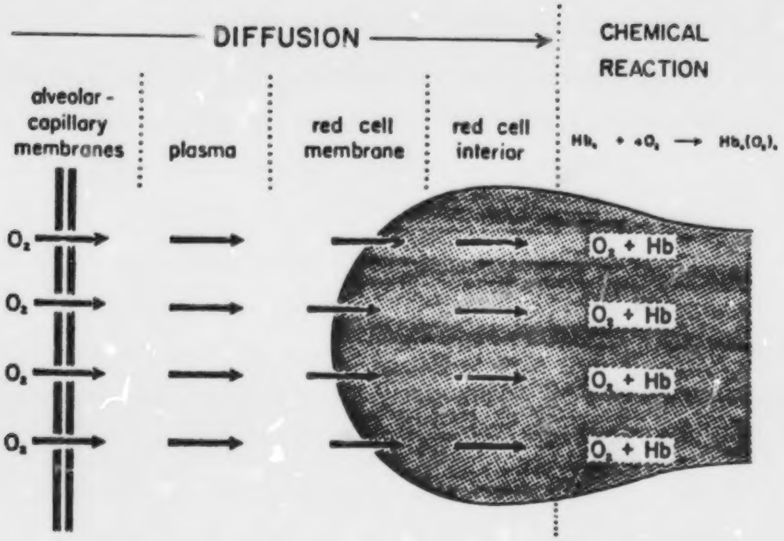

DIFFUSION
CHEMICAL REACTION
alveolar - capillary membranes
plasma
red cell membrane
red cell interior
Hb₄ + 4O₂ → Hb₄(O₂)₄
O₂
O₂
O₂
O₂
O₂ + Hb
O₂ + Hb
O₂ + Hb
O₂ + Hb

FIGURE 7

Schematic representation of passage of O_2 molecules from alveolar gas to pulmonary capillary blood

(Source: Reference 17)

Pulmonary function tests provide objective information, including:

1) functional impairment; its presence or absence
2) the nature of the functional impairment; objective or restrictive type
3) the severity of the functional impairment
4) whether the degree of functional impairment is compatible with the disability claimed

To study the lungs and their ability to move air in and out, the respirometer was developed to measure the volumes of air inspired and expired. It consists of a drum which moves freely when air is moved in or out of the spirometer and which registers the volume of air on a graph. In this way, the total amount of air inspired can be measured (total lung volume or

TLV) and the tidal volume (approximately 500 ml) is demonstrated when the subject breathes in and out quietly during normal respiration. The volumes measured can show changes in disease states as, for example, in emphysema where the total lung volume is markedly increased.

Usually, when testing lung function, the subject expires air forcibly into the spirometer, which provides a measure called the forced expiratory volume (FEV) or forced vital capacity (FVC). Usually, the maximum amount of air forcibly expired in one second (FEV_1) is measured.

In this way, the rate at which a given amount of air is moved out of the airways is measured, providing diagnostic information of pulmonary disease. For example, a worker exposed to enzymes in laundry products will show an FEV_1 markedly decreased during the working day compared to his normal FEV_1 measured during leisure hours (18).

A more complex spirometer is used to measure more subtle variations in flow rates. These are referred to as wedge spirometers and are attached to an X-Y recorder which graphically records air volume against flow rate (Figures 8 and 9) as the subject forcibly expires into the spirometer's mouthpiece.

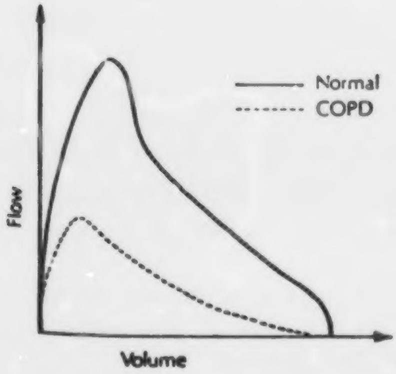


FIGURE 8

Schematic representation of a flow-volume curve. note how the size and shape of the curve is dramatically altered by obstruction, i.e. COPD (chronic obstructive pulmonary disease).

(Source: Reference 19)

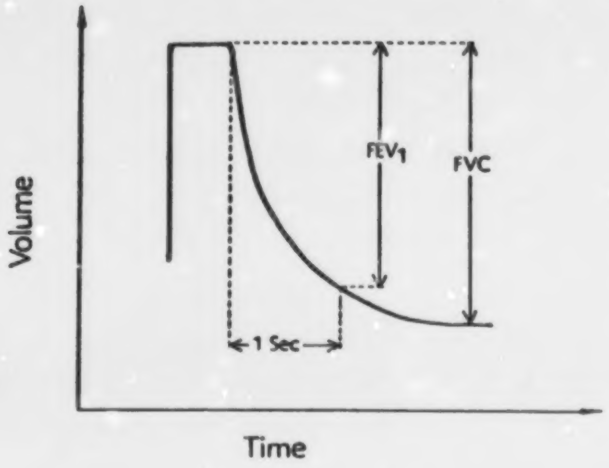


FIGURE 9

Schematic representation of a forced expiratory spirogram.

(Source: Reference 19)

Flow rates can be measured at 50% of the lung's vital capacity or volume, and hence information on the flow rates in the larger airways can be gathered. The flow rate measured at 25% of the volume (V_{25}) gives the physician information about decreased flow rates in small airways of 2 mm in diameter or less. This condition is seen in the pack-a-day smoker or in the worker who has developed an asthma condition over a long period of time.

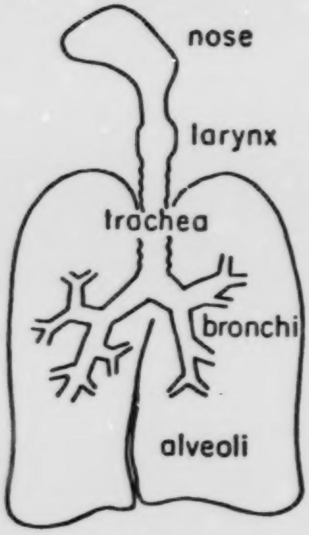
Many subjects with reduced flow rates have increased airways resistance (indicated as R_{aw}). The usual method to measure this value is to use a body plethysmograph which is a large box in which the subject sits. An air-tight door is closed and the subject breathes the air in the box through a mouthpiece. At the end of expiration, the mouthpiece is occluded which forces the subject to continue to breathe against a shutter which acts as an obstruction. Changes in pressure are recorded (ΔP), as well as changes in volume (ΔV), which permits calcuation of the R_{aw}, according to the following relationship:

$$R_{aw} = \frac{\Delta P}{\Delta V}$$

Subjects who suffer from obstructive lung disease, which includes smokers and those with bronchitis, usually show a marked increase in airways resistance. Because of the large bulky equipment and the highly delicate, calibrated meters used, this valuable test can rarely be done in the occupational field, such as at a mine site (17).

In the occupational world, the human being is an embattled fortress; embattled because the environment contains elements that can prove toxic to the human and a fortress because the body has many defenses to meet the challenges.

The human integument, that organ made of up skin and hair is relatively impermeable to low levels of substances. The orifices to the body, including the eyes, ears and mouth have their own special defenses. The lung and respiratory passages also have their protective devices (20) and Figure 10 shows a diagram, in a compartmentalized fashion, of the respiratory defenses and disease states which result from alterations in defense mechanisms. Table 3 provides additional details of the respiratory tract defense mechanisms.



COMPARTMENT	DEPOSITION	CLEARANCE	PATHOLOGY
NP Nasopharyngeal	impaction diffusion interception electrostatic	muco-ciliary (minutes) sneezing blowing	inflamation ulceration cancer
TB Tracheobronchial	impaction sedimentation diffusion interception	muco-ciliary (hours, longer?) coughing	broncho-spasm obstruction cancer
P Pulmonary	sedimentation diffusion interception	solubilization phagocytosis interstitial (hours to years)	inflamation edema emphysema fibrosis cancer

FIGURE 10

Compartmental Model of the Respiratory Tract

(Source: Reference 10)

TABLE 3

Mechanisms that Contribute to the Defense of the Respiratory Tract

NONSPECIFIC DEFENSE MECHANISMS

- **Clearance**
  - Nasal clearance
  - Tracheobronchial clearance
  - Alveolar clearance
- **Secretions**
  - Tracheobronchial lining (mucous)
  - Alveolar lining (surfactant)
  - Lysozyme
  - Interferon
  - Complement
- **Cellular defenses**
  - Nonphagocytic
    - Airway epithelium
    - Terminal respiratory epithelium
  - Phagocytic
    - Blood phagocytes (polymorphonuclear, neutrophilic leukocytes, monocytes)
    - Tissue phagocytes (alveolar macrophages)

SPECIFIC DEFENSE (IMMUNOLOGIC) MECHANISMS

- **Antibody-mediated (B lymphocyte-dependent) immunologic responses**
  - Serum immunoglobulins
  - Secretory immunoglobulins
- **Cell-mediated (T lymphocyte-dependent) immunologic responses**
  - Lymphokine-mediated
  - Direct cellular cytotoxicity

(Source: Reference 450)

The first line of defense is an aerodynamic one. Hairs in the nose, irregularly structured nasal walls and sticky mucous allow large particles to impact and be retained on the walls of the nose and upper airways. Also, the inhaled air is very turbulent in the trachea and this turbulence continues as smaller bronchi keep dividing, so that particles continue impacting in the larger airways.

Thus, air reaching the alveoli is free of suspended particulate or pollutants larger than 3 microns (u) in diameter. The relationship between particle size and the retention of respirable particles is given in Figure 11. It is of importance to note that small particles remain suspended in air for longer periods than large particles, thus increasing their hazard (Table 4).

The mucous lining in the bronchioles or air passages, to which particulate can stick, also removes some gases (Figure 12). Those gaseous pollutants with a high solubility in water, such as sulphur dioxide (SO_2) and chlorine, are largely removed by the superficial fluid in the mucosa of the airways. The smaller particles (about 1.5 u) and gases with lower water solubility reach the alveoli where respiration takes place.

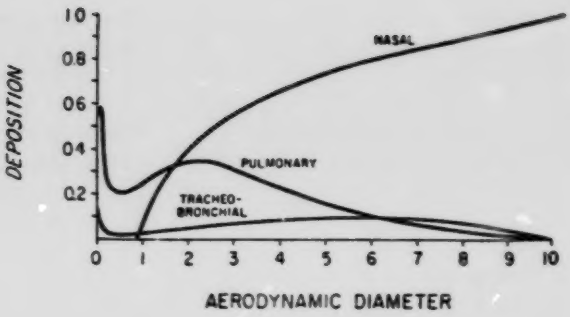


FIGURE 11

Nasal, tracheobronchial and pulmonary deposition as a function of particle size; breathing pattern, 15 breaths/min, 750 ml tidal volume

(Source: Reference 450)

TABLE 4

Rate of Fall of Spherical Particles in Still Air

Particle Diameter (u)	Rate of Fall (cm/sec)
100	30
10	0.3
1	0.003
0.1	0.00009

(Source: Reference 21)

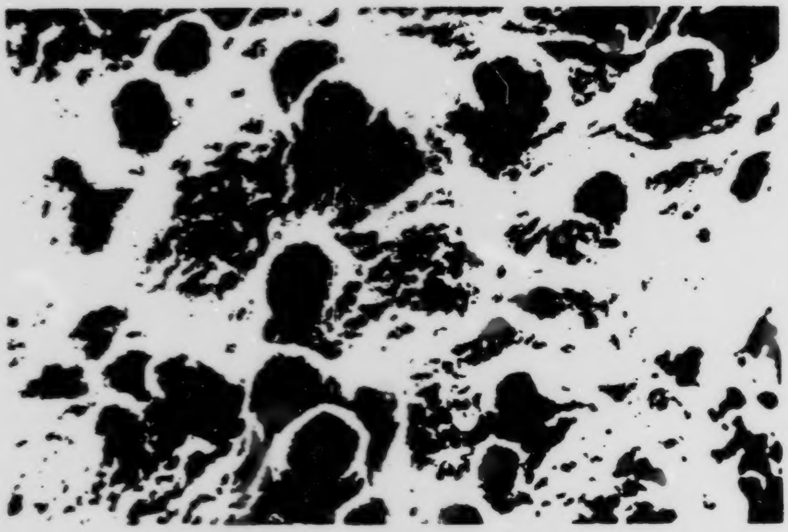


FIGURE 12

Scanning micrograph photo of mucous blanket lining a bronchiole

(From American Lung Association Bulletin, April, 1974)

As the particles impact on the mucous of the airways, they become part of an upward moving layer of fluid called the mucociliary escalator (Figures 13 - 16). This defence consists of small rods, called cilia, that beat upward and on which "floats" a layer of mucous. This effectively moves particles upward, where they are either swallowed into the gastrointestinal tract or expectorated.

The mucuous on the cilia usually remains at approximately 5 u in thickness. If it is increased to 25 u, the mucous stops moving. This occurs in individuals who have increased mucous production, as in chronic bronchitis and with many smokers, as well as following excessive exposure to sulphur dioxide (20).

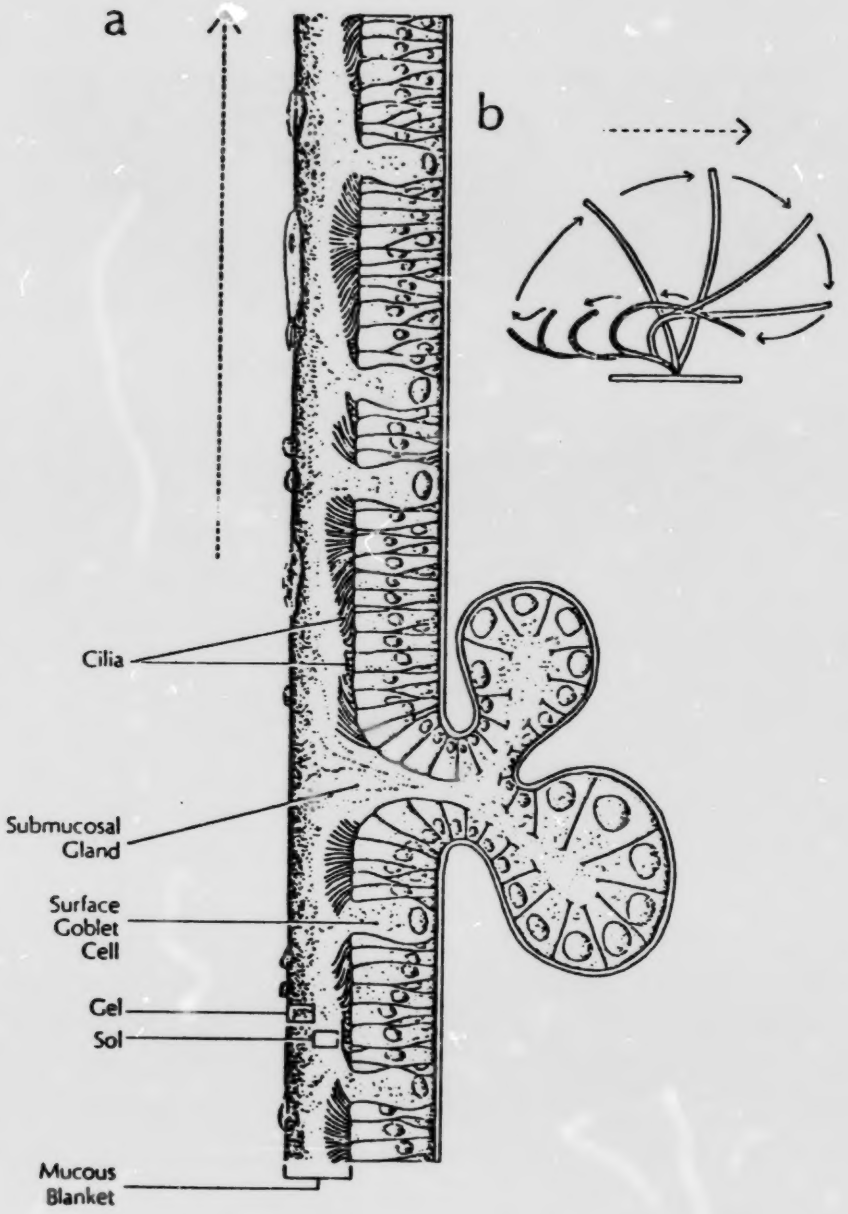

a
b
Cilia
Submucosal Gland
Surface Goblet Cell
Gel
Sol
Mucous Blanket

FIGURE 13

The mucociliary escalater. (b) is conceptual scheme of ciliary movement, allowing forward motion to move viscous gel layer and backward motion to take place entirely within more fluid sol layer

(Source: Reference 19)

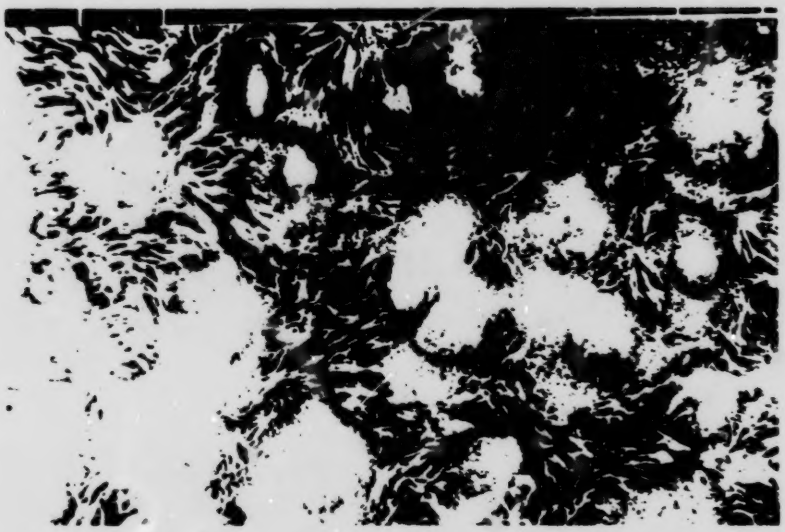


FIGURE 14

This scanning electron microscopic photograph shows the cilia after the mucuous blanket is removed. Interspersed among the ciliar are other bronchial-lined cells

(From American Lung Association Bulletin, April, 1974)

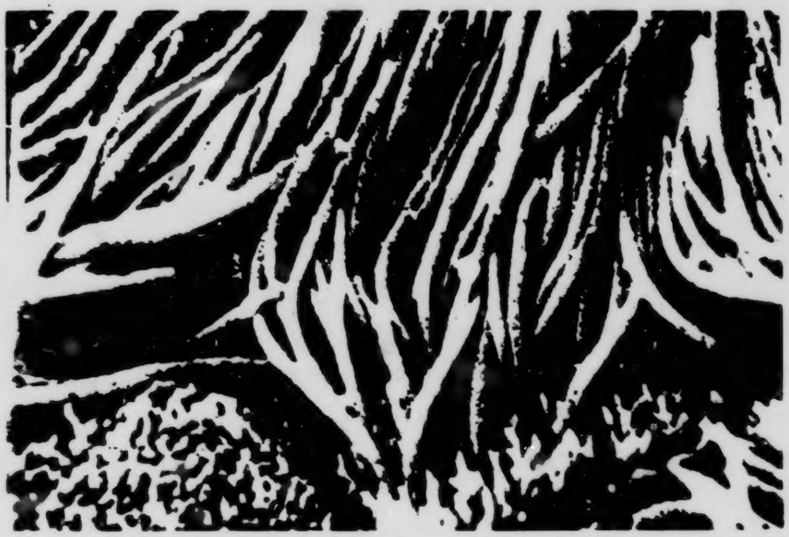


FIGURE 15

Scanning electron microscopic photograph of lung cilia at higher magnification. The round short "fibre"-looking structures at the lower left are microvilli, which transport fluids across the bronchial lining

(From American Lung Association Bulletin, April, 1974)

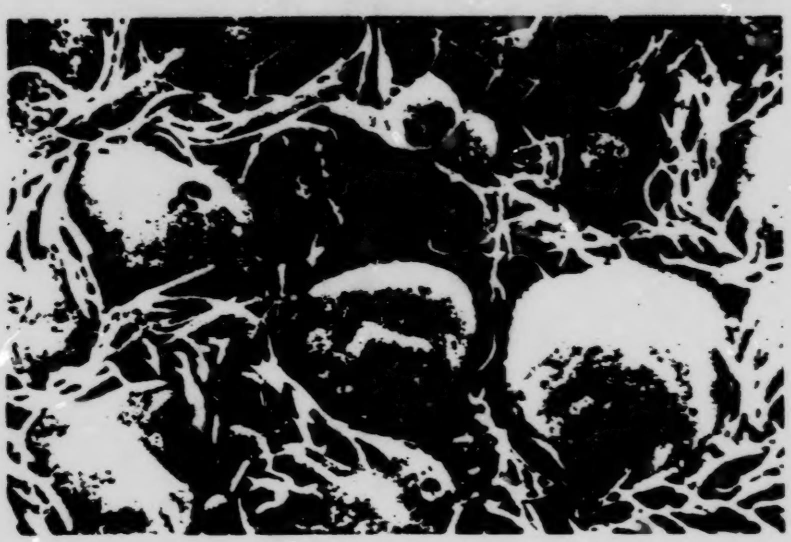


FIGURE 16

Scanning electron microscopic photograph of goblet cells found under the mucous blanket and which secrete the mucous

(From American Lung Association Bulletin, April, 1974)

Mechanisms of fluid transport in the alveolar portions of the lung are less clearly understood (452). Studies of particle transport at this level of the lung have shown that they move both centripetally to the bronchioles and centrifugally to the perivascular or subpleural areas. Whether this transport occurs on the surface of the respiratory membrane or within the interstitium is not clear. Although it seems clear that most particles are transported within phagocytes, it is not known whether those found in the interstitium penetrate as naked particles or are carried there by penetration of dust-laden phagocytes.

Green, in his review of the clearance of infectious agents from the pulmonary system (452) pointed out that particle clearance of the respiratory membrane occurs in two distinct phases. The initial phase is rapid with a half-life of about 100 hours. The second phase has a long biological half-time of 62 to 70 days, with some material remaining in the lung permanently. The rates of removal can be influenced by the type of material, its cytotoxic activity on the phagocytes, as well as the presence of lung disease or alterations in function.

Macrophages are another part of the pulmonary transport system which functions to remove objectionable pollutants. These are large cells that engulf or "eat up" particles penetrating to the alveoli. By ingestion, the particles are contained within the cells and prevented from harming the alveolar membrane. Macrophages eventually reach the mucociliary escalator and are carried out of the respiratory system. This is one of the most effective lung defence systems. It has been found that four days after a massive dust exposure the alveoli on the outer periphery of the lungs were cleared of dust (20, 22). Impressive scanning microscopic photographs, which clearly depict macrophage activity, have recently become available, and are reproduced in Figures 17 and 18. As will be seen later, the macrophage, which differentiates from the monocyte, is a cell of primary importance to the health of the pulmonary system exposed to diesel particulate. The macrophages are derived from the blood monocytes and are widely distributed in body tissues, especially lymph nodes, liver and spleen, etc. They are easily washed out of the lungs. Macrophages are usually a fairly large cell of 20 - 80 um in diameter, with phagocytic function and containing many lysosomes. They are mobile cells and exhibit chemotaxis or movement to sites of tissue injury from which certain chemicals are released.

Phagocytosis involves the engulfing of foreign particulate matter by way of a pseudopodia which develops from the macrophage (or histiocytes). Degradative enzymes in the lysosomes may then act on the entrapped material to digest it. Ingestion of non-digestible substances, such as silica, soot, etc., may interfere with lysosomal function or even result in their rupture and release of its contents.

Policard estimated that a French coal miner working thirty years underground would inhale 6 kg (13.2 pounds) of coal dust, but at autopsy, the lungs contained only 60 - 80 g (2 - 2.5 ounces) of dust. This suggests that pulmonary clearance is 98 to 99% effective. Similarly, it has been estimated that the resident of a typical English industrial town would respire a lifetime carbon intake of 100 g and yet only 0.5 to 1 g is found in the lungs and

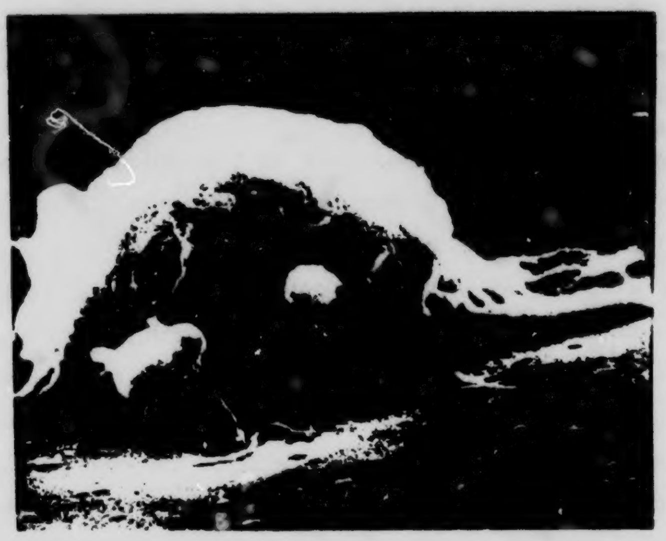


FIGURE 17

Scanning electron microscopic picture of a lung macrophage ingesting fly ash particles

(From American Lung Association Bulletin, April, 1974)

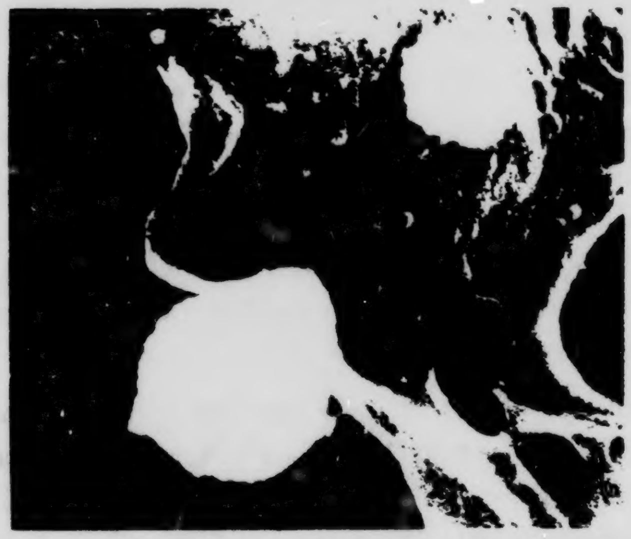


FIGURE 18

Close-up of a lung macrophage ingesting a fly ash particle

(From American Lung Association Bulletin, April, 1974)

bronchial glands at autopsy. However, a reduction in effectiveness of 1% doubles the dust load in the lung. It is inadequacies as small as 1 or 2% that lead to the development of disease, such as coal workers' pneumoconiosis.

If a pollutant particulate reaches the alveoli, there is another set of defences mobilized to protect the delicate respiratory membranes. If the insult is mild, as in the case of a non-fibrogenic dust, the alveoli will collapse, enclosing the dust. If the insult or injury is severe, as in the case of quartz dust, the alveolar lumen fills with tissue made up of macrophages, reticulin fibres and histiocytes, and as well, the alveoli and alveolar ducts collapse. Eventually, these latter developments lead to dense scar tissue formation which results in the lung tissue becoming useless for gas exchange. At the same time, the pollutant in the damaged lung cannot cause further damage to the lung parenchyma or tissue.

Two types of lung disease may develop, referred to as obstructive or restrictive in nature.

The obstructive disorders are most frequently encountered. The characteristic common to all obstructive disorders is an increase in the resistance to air flow through the pulmonary system. The reason for the reduction in airway flow can vary from condition to condition, and may be due to edema of the airways, fibrosis of the tubules with consequent narrowing, or may be due to bronchoconstriction in which the smooth muscles constrict and effectively reduce the airway diameters and thus increase resistance to air flow. Airway resistance can be of an acute or chronic nature and may be reversible or irreversible.

Dyspnea is a common feature of obstructive lung disorders. Patients with obstructive lung disease must work harder during breathing than normal in order to overcome the higher resistance to air flow through the airway passages. As well, there may be an uneven distribution of air into the lungs leading to regional differences in oxygen exchange at the alveoli, which can result in arterial hypoxemia (low oxygen concentration) and if severe enough, to carbon dioxide retention and respiratory acidosis (due to an increase in the carbonic acid concentration of the blood).

Another feature of obstructive lung disease is that generally the volume of gas left in the lungs after a maximum expiration is more than normal, but as indicated above, the expiratory flow rates are usually depressed, with the total lung capacity being normal or above normal.

In advanced obstructive disease the chest may be enlarged, and is often termed barrel chest. Wheezing, generally during expiration, is not uncommon in obstructive lung disease. Obstructive lung disease can be serious enough that a patient can find it difficult to blow out a match by a rapid expiration of air because of the reduced flow of air through his pulmonary system.

With these common features in mind, the obstructive lung diseases can be categorized into two broad groups; (1) reversible conditions such as bronchial asthma and (2) irreversible conditions which persist or get progressively worse despite treatment and which include obstructive emphysema, chronic bronchitis and bronchiolitis.

The primary pulmonary change in restrictive lung disease is a decrease in the total lung capacity. In most restrictive diseases, there is an decrease in elastic recoil of the lung, resulting in what might be termed a "stiff" lung. The diseases are usually classified into two broad categories, pulmonary edema and chronic intrinsic restrictive lung disease.

Pulmonary edema is the major cause of acute restrictive lung disease. In this condition, water and solutes accumulate in the extravascular pulmonary spaces, thus physically increasing the elastic recoil of the lung. The etiology of pulmonary edema can be traced to one of 3 mechanisms;

1) increased pulmonary capillary pressure,
2) increased pulmonary capillary or alveolar epithelial cell permeability, and
3) a combination of (1) and (2).

Cardiac disease is the commonest cause of increased pulmonary capillary pressure which results in an increased pressure within the capillaries of the lungs and a loss of their normal tone. This leads to more fluid moving from the capillary into the interstitial space of the alveoli, filling it with extracellular fluid. If the pulmonary capillary pressure is chronically elevated, an inflammatory response may occur and produce chronic changes, including fibrosis of the pulmonary vessels and the interstitial space leading to a lung with increased elastic recoil.

If there are abnormal permeability characteristics of the pulmonary capillaries or the alveolar epithelium, acute or subacute pulmonary edema may develop. This condition is referred to as adult respiratory distress syndrome (ARDS). It is frequently found in patients whose lungs were initially normal but who have undergone a severe systemic illness. In well-established disease, the death rate may be as high as 50%.

The disease begins with injury to the pulmonary capillary endothelium or the alveolar epithelium. The injury results in a change in the permeability of either the capillaries or the alveolar epithelium. Increased permeability of pulmonary capillary endothelium results in an excessive loss of water and solutes from the pulmonary capillary bed, which accumulate in the interstitial space initially, and then within the cells, and finally in the intra-alveolar spaces. This exudate results in an inflammatory reaction which produces additional changes in the interstitial space, capillary endothelium and alveolar epithelium. Changes in the characteristics of the surfactant coating system in the lung may also occur which can result in alveolar instability and collapse. Atelectasis (airlessness of the lungs due to failure of expansion or resorption of air from the alveoli) is a prominent feature of ARDS.

Generally patients develop progressive tachypnea (rapid breathing), lung markings increase, as observed by X-ray, and eventually nodules develop in the reticular system. Eventually the lung may assume a white-out pattern with little air-bearing tissue visible. The lungs become stiffer as the disease advances. Arterial hypoxemia occurs early in the disease, which can be corrected by administration of air with oxygen. Arterial CO_2 tension is low and the blood pH tends to be high. Eventually, the patient will reach a degree of involvement where positive pressure breathing is required in order to maintain an adequate arterial PO_2 level. At the terminal stages of the disease the CO_2 tension rises so high that respiratory acidosis occurs, compounded with metabolic lactic acidosis and the patient succumbs to the disease.

There are over a dozen conditions which may lead to ARDS, including chemical and mechanical injury to the lung tissue, fat embolism, infection, shock, smoke and burns.

3.2 The Dose-Response Relationship in Toxicology and Carcinogenesis

Although it is not intended to present a treatise on general pharmacology, toxicology or carcinogenesis, some background information on the dose-response relationship, especially as it relates to the mechanism of cancer development, will assist the reader in understanding the complexity of the health implications of the miner's environmental life.

For most chemicals and drugs which have pharmacological or toxicological properties, the biological effects, as measured in animals, usually increase with increasing dose of the agent in a manner similar to that depicted in Figures 19 and 20. Figure 19 shows how the response to a substance decreases as the dose is decreased. At some dose level above zero there reaches a point, called the threshold dose, where the agent elicits no response in the animal. When setting safe limits of exposure to chemicals, the threshold dose, or the dose which just elicits a response is used, but a "safety factor" of 10 -100,000 or more is applied. The value of the "safety factor" used depends on the chemical, the severity or critical nature of the response, and the philosophy and/or attitude of the scientists conducting the research. The "safety" values applied are frequently arbitrary. This is, in effect, the manner in which threshold limit values (TLV's) have been established by the American Conference of Governmental and Industrial Hygienists (ACGIH), as well as government and corporate regulatory bodies.

As will be discussed in more detail later, the TLV's are particularly applicable to such substances as CO, SO_2, NO_2, dust, etc., and are considered, on the basis of scientific evidence, to be a valid approach to determining safe levels of exposure for workers. A summary review of TLV's pertinent to diesel emissions and the rationale upon which the TLV's were established can be found in References (5) and (23).

The situation is much more complex when considering cancer-causing compounds (carcinogens) than for other types of environmental toxicants.

First, there is no unanimity of opinion on whether the simple dose-response relationship illustrated in Figure 19 exists between cancer-inducing chemicals and the expression of the disease. This document is not an appropriate media for a detailed discussion of this controversial and complex topic, and those interested are referred to a number of articles where the issues are debated with supporting scientific data (25, 26, 27).

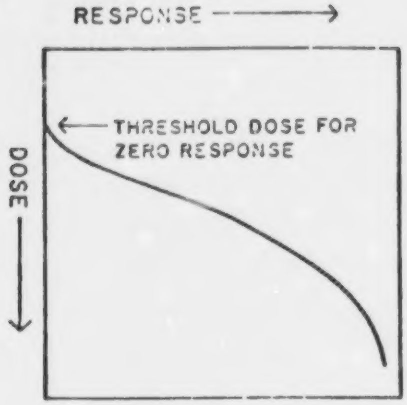

RESPONSE
THRESHOLD DOSE FOR ZERO RESPONSE
DOSE

FIGURE 19

Dose-response relation, showing graded downward change in response as magnitude of dose is reduced. A level of no response is reached at a threshold dose level above zero.

(Source: Reference 24)

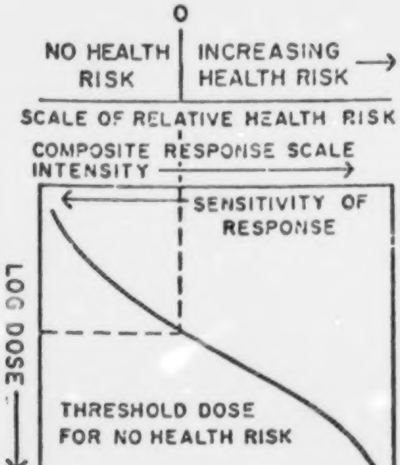

0
NO HEALTH RISK
INCREASING HEALTH RISK
SCALE OF RELATIVE HEALTH RISK
COMPOSITE RESPONSE SCALE
INTENSITY
SENSITIVITY OF RESPONSE
LOG DOSE
THRESHOLD DOSE FOR NO HEALTH RISK

FIGURE 20

Dose-response relation, assuming some kind and degree of response at all dose levels above zero. Derivation of the threshold dose is shown at the response level below which there is no significant health risk.

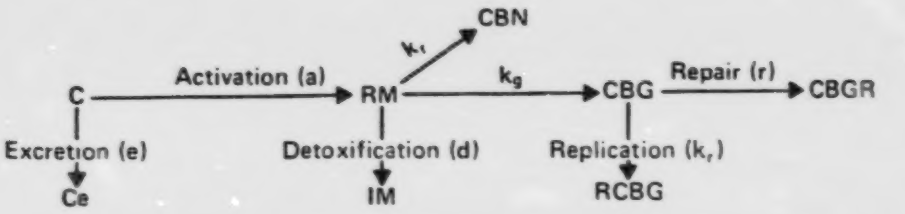
(Source: Reference 24)

The complexity of the mechanisms involved in chemical carcinogenesis, as it is presently understood, indicates even to the non-scientist, the degree of difficulty involved in resolving the question of whether there is or is not a pharmacological type dose-response relationship for carcinogens.

For example, cancer induction by a chemical carcinogen involves at least the following steps: exposure - absorption into the blood - distribution to tissues -activation - deactivation - elimination of the chemical and its metabolic products -binding to blood or cell constituents - interaction of the chemical with critical receptor sites - "injury" to cell chemicals and/or structure - repair of injury or proliferation of cells or cell death.

A simplified hypothetical model is reproduced in Figure 21.

Each of the processes or events indicated above may occur in a linear or non-linear fashion in relation to dose. That is, each step may or may not be dose-dependent. In some instances, a certain event or reaction may become saturated, thus allowing one or more of the reactants to be available for an alternate route of reaction, which may be more toxic than the saturated event itself.



C = Chemical
RM = Reactive Metabolite
Ce = Excreted Chemical
IM = Inactive Metabolite
CBN = Covalent Binding, Nongenetic
CBG = Covalent Binding, Genetic
CBGR = Repaired Covalently Bound Genetic Material
RCBG = Retained Genetic Program, Critical & Noncritical

FIGURE 21

Hypothetical model of the fate of a chemical carcinogen in the body requiring, for expression of carcinogenicity, activation to a reactive electrophilic metabolite and subsequent irreversible covalent reaction with a genetic receptor.

(Source: Reference 26)

In general, cancer-inducing chemicals cause a greater incidence of cancer when administered in larger doses than when given in smaller doses. Whether the dose-response curve is linear or non-linear may be debatable, but in either case, a dose-response relationship exists.

It is the present authors' opinion that there exists, for most carcinogenic chemicals, a dose below which the development of cancer is not likely to occur and/or there is a dose below which the risk of developing cancer is sufficiently low as to be acceptable to society. This concept is depicted in Figure 20, where it is shown that there is some degree of response at all dose levels above zero. However, below a certain dose, the diagram indicates there is no health risk.

In a recent review Nebert and co-workers (28) commented and concurred with the views of the present authors that if the dose of an environmental pollutant is plotted as a function of the percent of the individuals affected, and if some "effect" is measured, as either sub-threshold, threshold toxic or lethal, it is unlikely that effects would be found in 100% of the population receiving the same dose of the environmental pollutant. Rather, a multi-phasic curve would develop (Figure 22) in which the dose of the chemical causing toxicity in one person might be threshold, lethal or sub-threshold in other individuals. These authors explained this relationship as resulting from the complexity of the differing individuals genetic makeup in any given population. As examples, they noted that many pharmacological or toxic response to compounds such as cancer, mutagenesis, survival time, anemia, etc., can vary among siblings in the same family because of differences in drug metabolism between the individuals, which are regulated by a relatively small number of genes. The complexities of the metabolic fate of a chemical carcinogen, described in simplified terms in Figure 21 detailed by Nebert et al (28) can be reviewed in Figure 23. These authors presented a scheme demonstrating the relationship of a genetic locus in a mouse with carcinogenesis, mutagenesis, etc. (Figure 24).

The complexities of the interaction between environmental factors and the metabolism of chemicals and their relationship to the genetic makeup of individuals, was further reviewed by Vesell (29).

Unfortunately, quantitative data relating the dose of carcinogenic compounds to which miners might be exposed is sparse. In addition, the

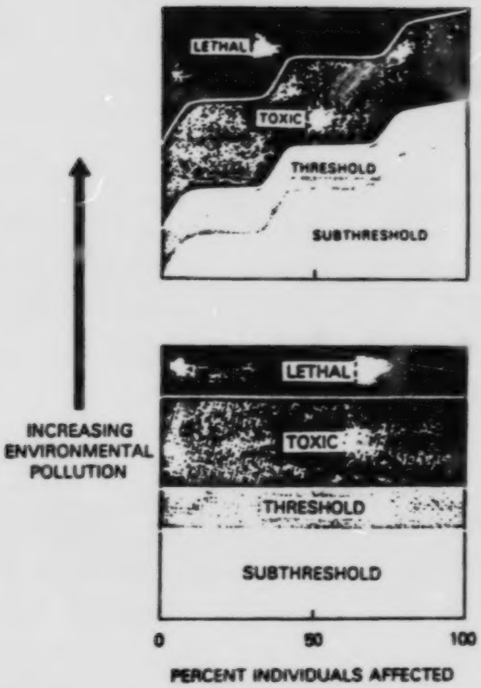

LETHAL
TOXIC
THRESHOLD
SUBTHRESHOLD
LETHAL
TOXIC
THRESHOLD
SUBTHRESHOLD
INCREASING ENVIRONMENTAL POLLUTION
0
50
100
PERCENT INDIVIDUALS AFFECTED

FIGURE 22

Hypothetic scheme depicting 2 experimental possibilities when a population is exposed to increasing amounts of environmental pollutants

(Source: Reference 28)

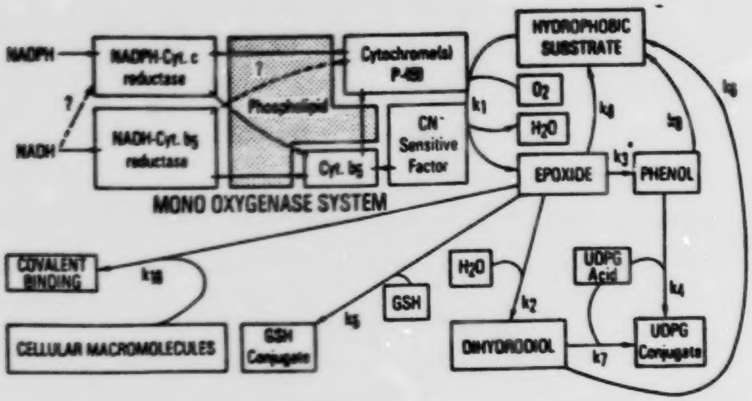

NADPH
NADPH-Cyt. c reductase
?
NADH
NADH-Cyt. b5 reductase
Phospholipid
?
Cytochrome(s) P-450
Cyt. b5
CN⁻ Sensitive Factor
MONO OXYGENASE SYSTEM
HYDROPHOBIC SUBSTRATE
O2
H2O
k1
k8
k9
k6
k3
EPOXIDE
PHENOL
COVALENT BINDING
k10
CELLULAR MACROMOLECULES
GSH Conjugate
k5
GSH
H2O
k2
DIHYDRODIOL
UDPG Acid
k4
UDPG Conjugate
k7

FIGURE 23

Scheme for the membrane-bound multicomponent monooxygenase system(s) and the various possibly important pathways for hydrophobic substrates. The reactive arene oxides or epoxides can rearrange nonenzymically to form phenols (or alcohols), be hydrated to form trans-dihydrodiols, be conjugated with glutathione, or be reduced back to the parent compound. For any given substrate, the relative rates of k_1 through k_{10} are currently not known and most likely vary among different tissues, strains and species. Age, nutrition, hormonal balance, diurnal variations, pH, and saturating versus nonsaturating conditions all possibly may be important factors in affecting these various rates.

(Source: Reference 28)

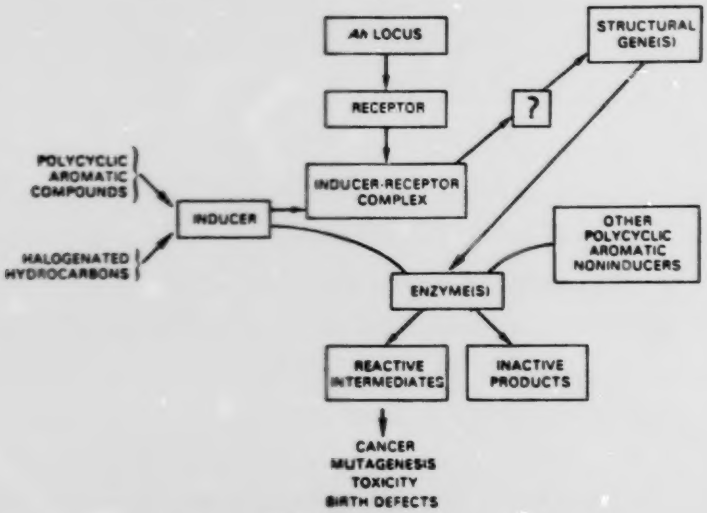

Ah LOCUS
RECEPTOR
STRUCTURAL GENE(S)
?
POLYCYCLIC AROMATIC COMPOUNDS
INDUCER
INDUCER-RECEPTOR COMPLEX
HALOGENATED HYDROCARBONS
OTHER POLYCYCLIC AROMATIC NONINDUCERS
ENZYME(S)
REACTIVE INTERMEDIATES
INACTIVE PRODUCTS
CANCER
MUTAGENESIS
TOXICITY
BIRTH DEFECTS

FIGURE 24

Simplified scheme demonstrating the relationship fo the Ah locus in the mouse with carcinogenesis, mutagenesis, toxicity and birth defects caused by environmental chemicals.

(Source: Reference 28)

correlation of the dose-response relationships found using animal-models are not always directly applicable to humans. For a considered opinion and elaboration of this most important topic, the reader is referred to References (27) and (30). Until additional scientific data are available which delineates the relationship between the effect of a specific carcinogen in animals and in man, animal studies provide the best estimate of risk for human exposure and must be used to suggest safe contaminant levels in the workplace.

Into this already complex and unresolved area we must add the plight of the miner who is exposed to radioactive elements. The most obvious relevant scene for Canada would be the uranium miner.

A great deal of experimental work on the induction of cancer by radiation has failed to clearly demonstrate whether there is a threshold or non-threshold dose-response relationship. Some investigators use radiation-induced cancer dose-response relationships to support the concept that there is no threshold dose for any carcinogen. However, as was pointed out by Gehring and Blau (26), it may not be valid to equate chemical carcinogenesis with radiation-induced carcinogenesis.

In the case of radiation-induced cancer, the radioactive rays or particles penetrate cells, release energy within the cells which results in the formation of free-radical molecules capable of interacting with chemical constituents of the cells in a manner which may eventually lead to abnormal cell proliferation or cancer.

The series of events which occur after radiation exposure are described by physical-chemical laws and theoretically only one radioactive particle is required to initiate a cancerous response. Although after radiation injury a complex series of events may occur in which a cell may repair or not repair, leading to a normal or abnormal cell, respectively, the overall events leading to radiation-induced cancer seem simpler than for chemically-induced cancer. For example, in the latter case, many activation-deactivation events may intercede between the time of exposure and the expression of the disease, which are not operational in radiation-induced malignancy.

However, dose-response relationships have been described, and doses below which the risk of developing cancer are negligible have been determined.

To this involved story of actions and interactions must be added the additional points that changes in physiological state, age, sex, hormonal status, nutrition, exposure to concomitant chemicals etc., can modify or increase the response to a particular carcinogen.

The miner is exposed to a particularly complex environment. He may encounter particulate matter from both rock and diesel exhaust, gaseous diesel emissions, including SO_2, CO and NO_X, as well as various aromatic constituents, including aldehydes, ketones and hydrocarbons, in addition to carcinogenic polynuclear aromatic compounds (PNA's), such as benz(a)pyrene. Add to this the presence of oil and acid mists, high humidity, blasting fumes, noise, vibration and thermal stress, all of which can effect the physiological and mechanical status of the miner's respiratory system, as well as gamma (γ) and alpha (α) radiation from the decay of uranium and other radioactive constituents and the complexity of the miner's environment is readily apparent. However, this does not infer that the mining environment must be or is necessarily unsafe from a health point of view. Cautious and judicious concern is, however, obviously in order.

To assess only the toxicity of individual components and not to estimate the total effect due to the interaction of the entire plethora of substances would be irresponsible and unscientific. However, data are not available which permit reasonable conclusions to be made concerning the total toxic potential of the miner's environment. Therefore, risk estimates must be calculated which will permit defensive actions to be taken until such time as quantitative data are available which will permit scientifically defensible decisions to be made relative to protecting the miner's health and well-being.

There are many examples in the literature which clearly demonstrate the complexity of carcinogenesis resulting from the ingestion or the administration of various carcinogenic agents. Additive, and even synergistic effects have been described where one carcinogen, or even a non-carcinogen, has resulted in an increase in tumour formation when administered together with a second chemical carcinogen.

From a review of the literature it appears that, in order to adequately protect the health of workers, one must consider that carcinogenesis can result from the overall impact and additive effects of many chemicals to which the miner may be exposed (31, 32, 33, 34, 35, 36). This approach and philosophy has been used in the development of the recommendations given in this report.

The authors realize and fully understand the pragmatism of the situation. Mining is an important economic reality to corporations, governments, and the miners themselves. No matter what deleterious effects the mining environment may have on the workers, the industry must and will remain viable. The authors have kept this in mind throughout the preparation of this document. When data were not available to permit definitive conclusions relative to the toxicity of substances or combinations of substances, then estimates of risk have been made which hopefully will permit the miner to appreciate his exposure and related health implications and permit him to make appropriate decisions relative to his employment as a miner.

Relative to this discussion are the comments of Moll and Tihansky (37) that "most regulatory agencies as well as researchers lack an appreciation of the complexities underlying benefit estimation. As a result they tend to establish controls which minimize risks without explicitly considering product benefits. "Zero-tolerance laws", which prohibit any detectable amount of a hazardous material, are inflexible and can quickly become too costly". These authors went on to discuss methods of making risk-benefit analysis for hazardous materials in the environment. They suggest a multiple-criteria approach involving the consideration of many factors bearing on the decision, in addition to those of "risk" and "benefit", such as "minimum reducible risk", maximum socially acceptable risk, maximum acceptable cost, and value of human life.

3.3 Diesel Engines

3.3.1 Mechanics and Chemistry

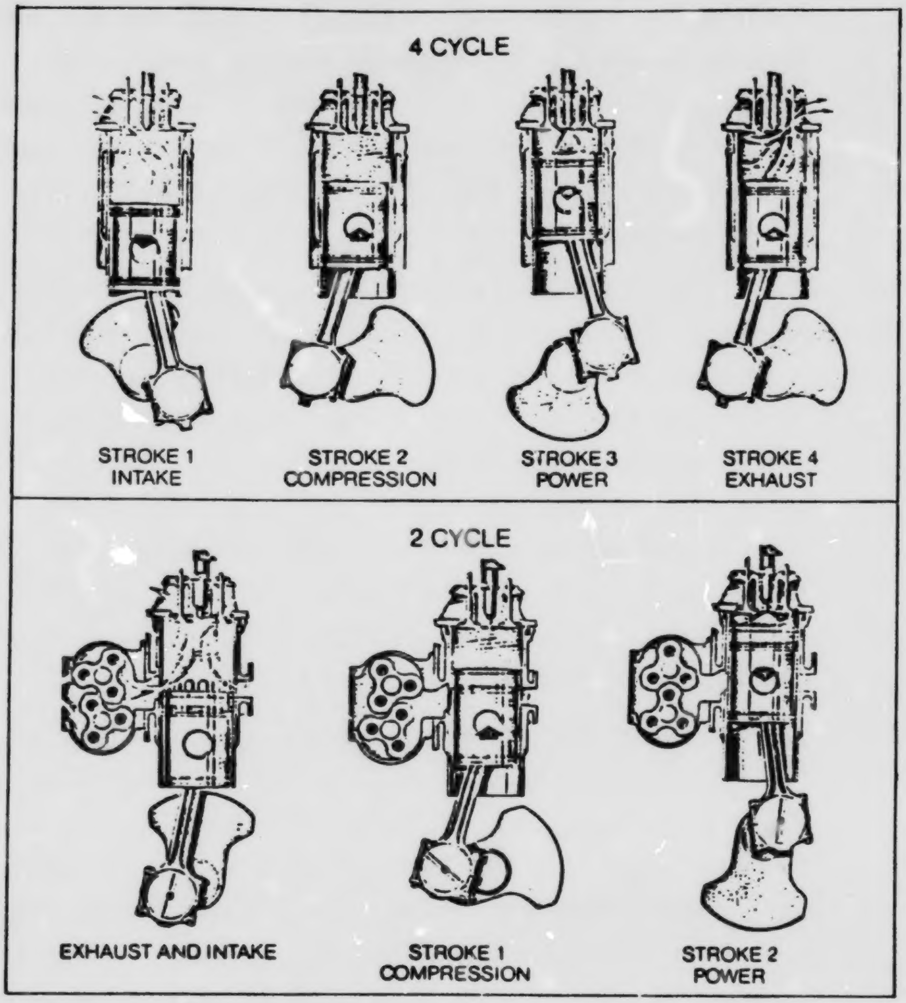
It is the intention of the authors to present sufficient background information related to the mechanics and operation of diesel engines to assist the reader in understanding the basic characteristics of diesel emissions. For more detailed reviews of diesel engine design and function, the reader is referred to References (12, 38 and 39).

There are two basic diesel engine designs - direct injection (DI), and indirect injection (IDI). Although differing in many respects, both have the common feature of being compression ignition engines, as compared to the spark ignition used with gasoline engines. Because of the relatively high compression ratio (15 - 20:1), the lean fuel (high air-to-fuel) mixtures and the different fuel used, as well as other features, the components of diesel exhaust differ markedly from those of gasoline engines. The power output of a diesel engine is dependent on the quantity of fuel injected into the cylinder, which can be varied while the amount of air entering the cylinder remains constant.

In the DI engine, fuel is injected under high pressure into a depression on top of the cylinder and the piston rises during the compression stroke. With the IDI diesel, or prechamber type, the fuel is first injected into a special chamber separated from the main piston. The prechamber is designed so that air is taken into it as the piston rises, causing it to "swirl" within the chamber. Self-ignition (due to the higher temperature resulting from the increased pressure) occurs in the prechamber which pushes the air-fuel mixture into the main chamber where further vaporization, mixing and combustion of the fuel-air mixture occurs.

If either type of engine uses ambient air, it is referred to as a "naturally-aspirated" engine. If air is introduced under pressure, the engine is of a "turbo-charged" variety. The latter design can increase the power output of an engine as much as 50%. If the air between the turbo-charger compressor and the engine inlet is cooled, a technique referred to as "after-cooling", the power output of an engine can be increased as much as 100% when compared to a naturally-aspirated engine.

Diesel engines have combustion cycles of intake, compression, power and exhaust. Two different power cycles can be used. A "4-stroke" or "4-cycle" engine uses four piston strokes and two revolutions of the crank shaft to complete a cycle. A "2-cycle" diesel completes the cycles in two strokes of the piston and one revolution of the crank shaft. Figure 25 shows these differences between the two types of engines.



4 CYCLE
STROKE 1
INTAKE
STROKE 2
COMPRESSION
STROKE 3
POWER
STROKE 4
EXHAUST
2 CYCLE
EXHAUST AND INTAKE
STROKE 1
COMPRESSION
STROKE 2
POWER

FIGURE 25

Diesel Engine Cycles

(Source: Reference 40)

Two-cycle engines are used with DI chambers only, whereas 4-cycle power systems are used with both DI and IDI engines. Although more common with 4-cycle diesels, both types can be turbo-charged.

These differences in engine design and function result in differing emissions characteristics, as well as differing engine performance.

The ratio of the amount of fuel and air (fuel:air ratio or F:A ratio) delivered to the cylinder affects engine performance, as well as the quantitative and qualitative characteristics of the emissions. The chemically-correct F:A ratio for most engines is considered to be near 0.068. This value refers to a combustion mixture containing 0.068 pounds of fuel for each pound of air.

A word of caution. Some publications refer to an air:fuel ratio, which is the reciprocal of the F:A ratio. The chemically-correct air:fuel value is 15:1.

Generally, for diesels employed underground, the F:A ratio is adjusted to a maximum of about 0.04 - 0.05 so that an excess of air to fuel is present at all times. This results in lower noxious emissions, especially of smoke, which occurs at higher F:A ratios. This adjustment is commonly referred to as "derating" the engine. When a low F:A ratio is used, the mixture is referred to as "lean", whereas with a high F:A ratio, the mixture is considered "rich".

If the combustion of one pound of diesel fuel with 15 pounds of air was perfect, about 225 cubic feet of exhaust products would be produced with a composition as indicated in Table 5.

However, combustion is not perfect and several components considered as pollutants are emitted, as shown in Table 6.

In Table 6, "others" includes chemically-altered and unaltered fuel constituents such as PNA's aldehydes such as formaldehydes, and acrolein, etc.

TABLE 5

Exhaust Products of the Perfect Combustion of Diesel Fuel

Nitrogen	73%
Carbon dioxide and oxygen	13%
Water	13%

(Source: Reference 38)

TABLE 6

Products of "Imperfect" Diesel Combustion

Hydrocarbon (HC)	1%
Carbon monoxide (CO)	1%
Nitric oxides (NO, NO_2)	1%
Carbon or smoke	1%
Sulphur dioxide	1%
Others	1%

(Source: Reference 38)

- The amount of air taken into a diesel engine is constant while the quantity of fuel varies with the work or load put on the engine. Thus, the composition of the exhaust emitted will vary throughout the work cycle, during which the load demand on the engine is changing. These points must be taken into consideration when discussing engine exhaust composition. For example, at low load or at engine idle, the F:A ratio will be lower than at higher loads and/or rpm's.

Before proceeding further with this general discussion on diesel engines, definition of some common terms is in order:

Rated Speed:

The maximum speed the engine is designed to run at - this is generally in the range of 1800 - 2200 rpm for diesel engines used in mines. This is also the speed at which the engine generates its maximum or "rated" power output.

Peak Torque Speed:

This is an intermediate speed and is the speed at which the engine produces maximum torque.

Idle Speed:

This is the speed of the engine when operating without a load and is usually around 600 rpm.

As pointed out by Stewart and co-workers (41, 42), a significant percentage of the torque developed by LHD diesel equipment, even at idle speed, is used to operate ancillary equipment such as hydraulic systems and converter pumps. It was found using a Deutz F6L-714 diesel-equipped LHD machine that as much as 42.7% of the available engine torque at high speed was required to rotate the engine-driven ancillaries. This led the investigators to conclude that the engine would be operating at or near maximum load for most of a normal duty cycle. The only segment which would be at much less than full load is the engine-leading return to the draw point (see Figure 26).

This point is emphasized because most of the engine emission data available in the literature has been obtained using laboratory engine-dynamometer set-ups in which torque is zero at idle speed. Obviously, such emission data would not necessarily be directly related to the output of LHD equipment as used underground. This is not to suggest that laboratory findings are not useful, but to recommend caution in the interpretation of such data when relating them to the underground situation.

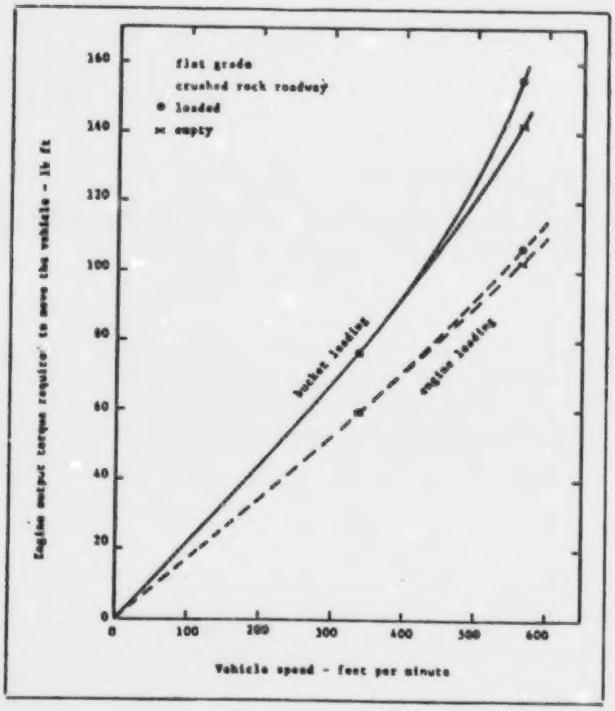


FIGURE 26

Variation in engine output torque required to move the machine versus vehicle speed for flat-grade hauling

(Source: Reference 42)

3.3.2 Measurement of Diesel Exhaust Components

Key to the issue of pollutant levels in exhaust are the validity of the analytical methods used to measure the volatile and non-volatile constituents.

To carry out a critical assessment of the many methods used, and/or published in the literature, is beyond the scope of this report and the capabilities of its authors. Johnson et al (43, 44) reviewed current methodology and detailed the limitations of the various instruments available.

Carlson and Johnson (9) provided a comprehensive review of the availability of continuous, real time, battery-operated portable instruments and recording devices in monitoring the mining environment. As well, they described the results of measurements in three production mines, White Pine Mine (copper), the Saint Joe Minerals Brushy Creek Mine (lead) and the Colorado Westmoreland Orchard Valley Mine (coal). These papers provide excellent background sources and update the problem of measuring diesel exhaust components in mine atmospheres. They emphasized the fact that although the measurement of exhaust concentrations of contaminants is useful, it is the total output which determines how much ventilation will actually be required to control the mine ambient air concentration of diesel pollutants. The total amount of any pollutant may be expressed in terms of amount per unit time, amount per unit of fuel consumed, or amount per unit of work performed. When expressed as g/BHP-hour, it is in terms of the rate of emissions divided by the rate of doing work by the engine, which the authors considered to be a useful expression when comparing emission rates between engines.

They noted that in the United States recently proposed Federal regulations for coal mine ambient air quality would adopt recommendations of the National Institute for Occupational Safety and Health (NIOSH) instead of ACGIH TLV's. The present TLV standards for exposure to gaseous contaminants and the Federal standard for coal dust are listed in Table 7.

TABLE 7

Present ACGIH TLV's for CO, CO_2, NO and NO_2 and Dust and Proposed Specifications for These Substances in Atmospheres of Underground Coal Mines

	Present Standard (1977 ACGIH TLV)			Change proposed for 30 CFR part 70.700 from 11/16/77 Federal Register		
Substance	**TWA[1]**	**STEL[2]**	**C[3]**	**TWA**	**STEL**	**C**
CO	50 ppm	400 ppm	--	35 ppm	--	200 ppm
CO_2	0.5%	1.5%	--	--	--	--
NO	25 ppm	35 ppm	--	--	--	--
NO_2	5 ppm	--	5 ppm*	1 ppm	--	1 ppm*
Dust	2 mg/m^3** respirable (coal mines)					
	10 mg/m^3*** total (metal-nonmetal mines)					

---

* Since no time limit is given the ceiling limit must never be exceeded.

** If greater than 5% quartz, the standard is 10% resp. quartz.

*** If greater than 1% quartz, the standard is set for the respirable fraction instead and is 10/(% resp. quartz).

1) TWA stands for time weighted average (8 or 10 hour shift)

2) STEL stands for short term exposure limit. It is to be interpreted as an absolute ceiling limit never to be exceeded. Excursions above the TWA up to the STEL are allowed for periods up to 15 minutes with at least 1 hour between such excursions.

3) C stands for absolute ceiling never to be exceeded.

Source: Reference 9

— The authors emphasized and exemplified the potential for erroneous conclusions relating to diesel emissions and the amount of dilution air required to provide safe levels of toxicants when comparing engines in the laboratory without knowing how they will be used in the field and they emphasized the need for field work to substantiate laboratory findings. They also noted that the consideration of a TLV below 5 ppm for NO_2 and a TLV for diesel particulate matter could completely change the future types of engines and control systems used in underground mines. This article also contained an excellent review of the special features peculiar to underground mines which present difficulties in monitoring air pollutants. It was considered that the most difficult place to ventilate in a mine is the dead end heading at the working face. The discussion reviewed the difficulty of using mobile monitoring equipment in a mine because of temperature variations, lack of electric power and relative humidities of 60 -100%. Obviously, special monitoring requirements are demanded in order to obtain real time values for mine environment contaminants.

The authors described in detail a mobile mine air monitoring laboratory and portable instrumentation used for the continuous monitoring of CO, CO_2, NO, NO_2, SO_2, NH_3 and particulate matter, as well as temperature and air velocity. These proliferative authors again indicated the utility of on-vehicle CO_2 measurements. Because 99% of the carbon in diesel fuel is emitted as CO_2, is stable in mine air, and its concentration is related to the degree of dilution resulting from ventilation, CO_2 is a useful component to measure. Also, the carbon dioxide concentration can be expected to be proportional to the concentration of other exhaust contaminants such as CO, NO, NO_2 and particulate matter. However, in contrast to carbon dioxide, the amounts of these other contaminants produced per pound of fuel consumed are markedly affected by the engine load and speed conditions. It appears that the best relationships are between CO_2 and the oxides of nitrogen, particularly NO, and diesel particulate matter. Those between CO_2 and CO exhibited a great deal of scatter. They suggested that a CO_2 concentration can be selected below which level the probability that the TLV's of the other pollutants would be exceeded could be relatively assured.

In describing instruments to measure the respirable particulate matter (RPM), it was pointed out that the RPM in the ambient mine air was derived from the ore itself, as well as from the diesel exhaust. These investigators developed a procedure for determining the percentage of the particulate matter which was of diesel origin based on a carbon-hydrogen-nitrogen analysis of respirable particulate matter collected on Gelman A/E glass fibre filters. It was found that the procedure was more accurate in mines where the ore contained virtually no organic matter than in mines where the ore dust contained over 10% carbon. Field and laboratory work indicated that samples containing from 0.2 -3.0 mg of carbon could be accurately analyzed with the equipment. In the two mines which were examined over 60% of the particulate matter was diesel-related. Descriptions were given of two rapid read-out, battery-powered, hand-carried continuous recording instruments, which provided a measurement of the respirable particulate matter in the mining environment. However, it was concluded that neither instrument gave reliable concentration measurements where the particulate matter was of diesel origin.

The authors concluded that when detector tubes (i.e. Drager) were used to monitor mine air where diesel equipment was in place, it must be recognized that the readings may be inaccurate because of variabilities in the manufacturing process of the tubes and because of the presence of interfering substances in the mine atmosphere. They reported errors considerably greater than those normally expected and the magnitude of the errors varied with the type of diesel engine and the manner in which it was used.

Table 8 indicates the instruments used in measuring the ambient air quality at the White Pine and Brushy Creek Mines (45).

A succinct summary of analytical methodology was derived at the 1977 - NIOSH Diesel Workshop. The emissions work group, comprised of twenty "expert" members, compiled the information on sampling and analyses reproduced in Table 9. The present authors feel this remains an up-to-date evaluation of pertinent methodology.

TABLE 8

Instruments Used to Gather Mine Air Quality Data at White Pine and Brushy Creek Mines during the Period 3/27/75 to 7/29/76

Instruments	Pollutants Measured by Instrument	Operating Principle	When and Where Used
Bartlesville Energy Research Center Small Sample Analysis System	CO, CO_2, CH_4 O_2	Chromatography Polarography	White Pine, 1975
Beckman 868	CO_2	Non-Dispersive Infrared (NDIR)	White Pine 1975, 1976
Ecolyzer 2600, Energetics Science Inc.	CO	Electrochemical	White Pine 1975, 1976
McMillan 2100	NO	Chemiluminescent	White Pine 1975, 1976
MSA 70	CO	Electrochemical	White Pine, 1976 Brushy Creek
Ecolyzer 7000	NO, NO_2	Electrochemical	Brushy Creek, Spring and Summer, 1976

Source: Reference 45

Table 9

STATE OF THE ART OF SAMPLING AND ANALYSIS OF DIESEL CONTAMINANTS

Analyte	Sampling Method(s)	Analysis (Current)	Reliability	Analysis on Horizon
CO	-----	NDIR	Good	
Particulate	Membrane filter Multistage impactor Air Dilution technique	Gravimetric Gravimetric Gravimetric	Good Good Good	Online - b scattering piezobalance
Polynuclear Aromatic Hydrocarbons	Membrane filter	Gas chromatographic (GC) Liquid chromatographic (LC) U.S. Fluorescence	Fair Fair Fair	Better derivatives and stationary phases
Unburned hydrocarbons	Absorption trap online	GLC, LC, hydrocarbon analyzer	Good	
Sulfur dioxide	Evacuated tubes (batch) online	West Gaeke strain length tube electrochemical Pulse fluorescence	Good Fair to Poor Good Good	
Sulfur trioxide (H_2SO_4)	Membrane filter impinger - liquid	Turbidmetric chloranillate	Fair	Flame photometric
Carbonyls	Impinger, absorption tube	General MBTH (spectro.) Acrolein GLC, stain tube HCHO GLC, stain tube	Fair Fair Fair	Better reagents and stationary phases
NO_X	Impinger	Phenoldisulfonic (NO_X) Saltzman (NO_2) NO_X and NO_2 Chemiluminescence(NO_X,NO_2) Electrochemical (NO_X,NO_2)	Good (high conc.) Good (low conc.) Fair to Poor EPA Ref. Method Fair	Newer generation of online
NO_2	Hot line	DNUV	Fair to Good	
Odour Components	Absorption tube Online	LC (A.D. Little) Odor Panel	Fair Fair	

(Source: 1977 - NIOSH Diesel Workshop)

3.3.3 Mass Emissions

Generally, the concentration of exhaust components is given in parts per million (ppm) or milligrams per cubic meter (mg/m^3). However, as stressed by Johnson et al (9, 45) total pollutant emission values are more important than concentrations in ambient air. It must be remembered that a larger engine puts out a greater total exhaust volume, and therefore a greater total pollutant output, than a smaller engine.

For example, the exhaust from two engines may both contain 1,000 ppm CO, but one engine has an exhaust flow of 5,000 cubic feet per hour (cu ft/hour) and the other 8,000 cu ft/hour. Obviously, the total amount of CO exhausted will be greater for the bigger engine. If the exhaust from both diesels is emitted into the same spacial area (for example, a mine heading), the concentration of CO in the area will be about 60% greater due to the emission from the larger than from the smaller engine. Thus, it is important that, for the underground situation, the concept of total or mass emissions be considered for any particular engine type.

Mass emissions may be calculated from concentration data and exhaust flow information as follows:

$$(HC)\text{ mass} = 0.0160 \times (HC)\text{ conc} \times M\text{ exh}$$

$$(CO)\text{ mass} = 0.0263 \times (CO)\text{ conc} \times M\text{ exh}$$

$$(NO_X)\text{ mass} = 0.0432 \times (NO_X)\text{ conc} \times M\text{ exh}$$

where:

(HC) mass, (CO) mass and (NO_X) mass are mass flow rates in grams/hour

(HC) conc, (CO) conc and (NO_X) conc are concentrations of each in the exhaust in ppm

and:

M exh is the mass flow rate of the exhaust pound-mass (lbm) per minute

where:

Mexh = M air + M fuel

and M air = air flow to engine, lbm/min

M fuel = fuel flow to engine, lb/min

In order to permit ready comparison of different engines, the emission rate is divided by the brake horsepower (bhp) produced by the engine and the resulting value is the brake-specific emission rate (BSHC). An example of the BSHC expression is:

$$\text{BSHC} = \frac{\text{rate of HC emission}}{\text{rate of doing work}} = \frac{\text{(HC) mass}}{\text{BHP}}$$

where: brake-specific hydrocarbon (HC) emission rate, grams/bhp-hr
and: brake power output, bhp

Holtz (39) published the relationship between two different engines at maximum and minimum power output shown in Figure 27. It should be noted that in both examples, the exhaust flow rate was more dependent on the speed of the engine (rpm) than on the power function. That is, the difference in exhaust volume at any given rpm did not vary greatly when the engine was operated at either full or low load.

Generally, mass emission data relative to diesel exhaust is expressed in terms of gram per brake horsepower hour (g/bhp-hr). Where applicable, this terminology will be retained throughout this report.

Caution must be exercised when discussing mass-emission values in g/bhp-hr, as they can represent somewhat misleading and inflated values. As an engine gets closer to idle, the NO output, for example, approaches zero. Although NO emission is significant (in terms of concentration or amount of NO per cu ft of exhaust), the horsepower is low, the exhaust volume is reduced and, therefore, the total absolute amount of NO emitted is lower than when the engine is working. The expression of emissions in g/bhp-hr is really only meaningful at higher loads or rpm's.

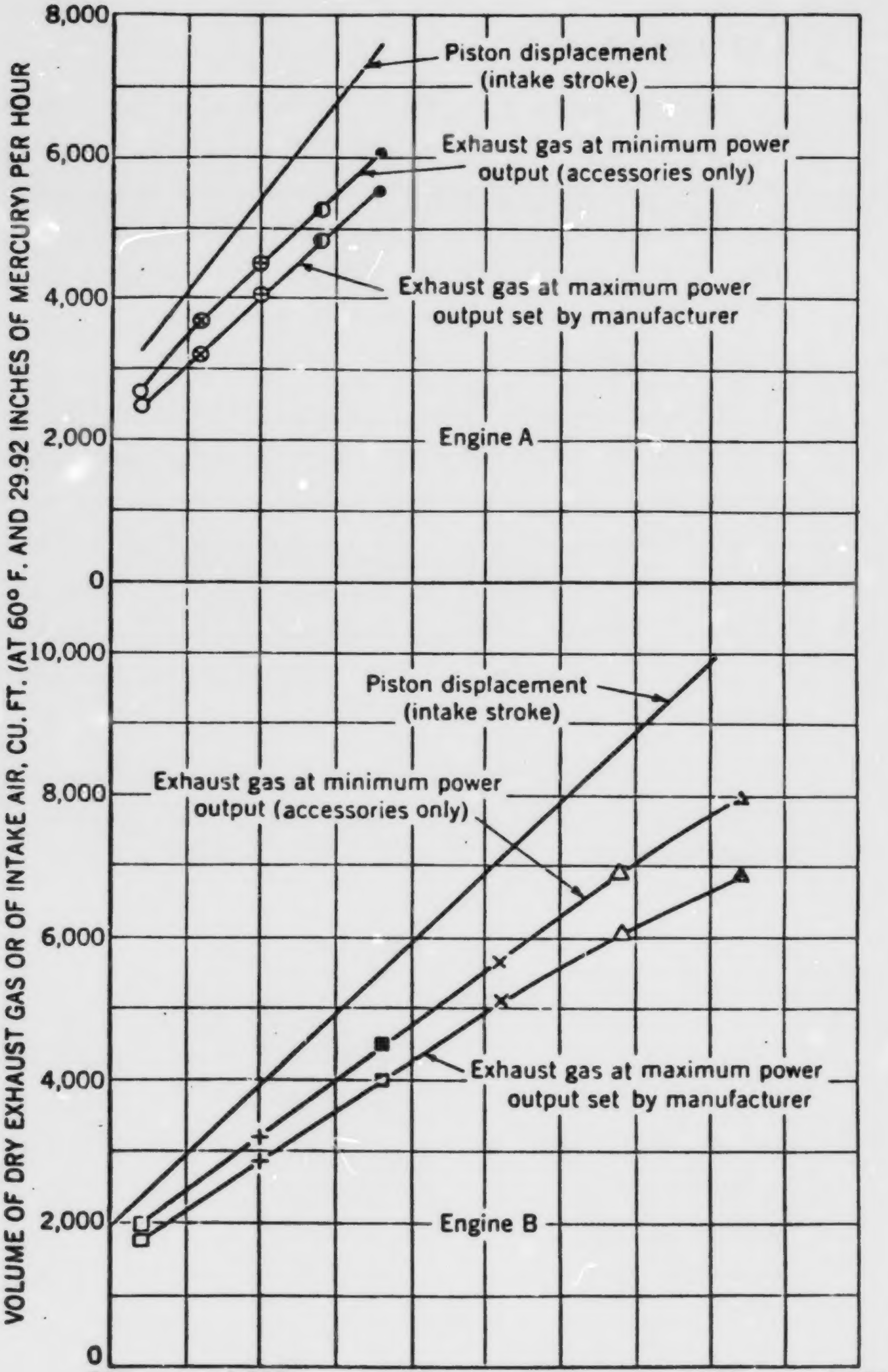


FIGURE 27

Relation of volume of diesel exhaust gas to engine speed at minimum and maximum power outputs

(Source: Reference 39)

3.3.4 Diesel Fuels

Diesel fuel is a petroleum distillate of a less volatile nature than gasoline. For use in underground equipment, most regulations require a fuel containing less than 0.5% sulphur. This results in lower SO_2 emissions, which is directly related to the sulphur content of the fuel. As will be discussed in detail later, this requirement takes on additional importance when engines are equipped with platinum catalytic converters.

There are three grades of diesel fuel available, commonly indicated as 1-D, 2-D and 4-D. The "highest" grade (lowest sulphur content) is 1-D and is the most volatile. Although the INCO metal mines in Sudbury and Denison Mines in Elliot Lake use 1-D fuel (1978), many mines, and most in the U.S.A., use 2-D. In the U.S.A., the Bureau of Mines requires that diesel fuel have a flash point greater than 140°F (60°C), which rules out the use of 1-D fuel, which has a flash point below this value.

Some typical characteristics of Number 1 and 2 diesel fuels are given in Table 10. Additional comparative information may be found in Reference (21).

TABLE 10

Characteristics of Diesel Fuels

	Number 1	Number 2
EP	565°F (296°C)	648°F (342°C)
Specific gravity (60°F)	0.814	0.835
Sulphur (% wt)	0.18	0.24 - 0.32
Viscosity (cs) 100°F)	1.66	2.5
Carbon/hydrogen ratio	6.20	6.41

Figure 28 shows the effect of air-to-fuel ratio on SO_2 emissions (proportional to percent load between 20:1 and 125:1, representing 100% and 0% load) for fuels containing various levels of sulphur. Further discussion of this aspect of diesel fuels will be found in later sections of the report.

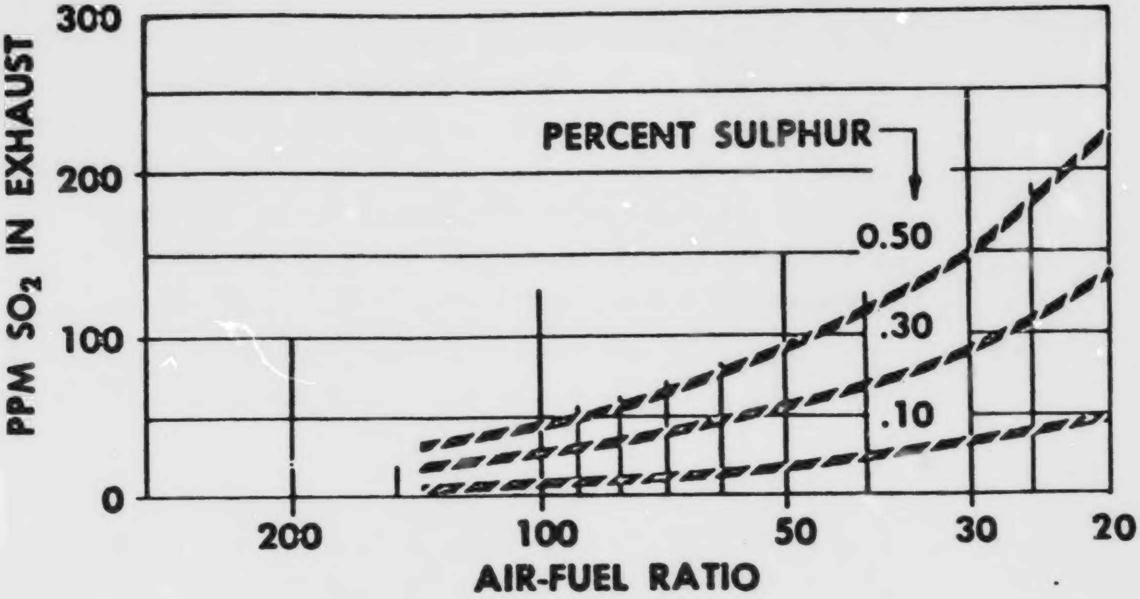


FIGURE 28

Effect of percent sulphur in diesel fuel on sulphur dioxide emissions assuming all sulphur is converted to SO_2

(Source: Reference 38)

Gibbons and Wolff (46) pointed out that in reciprocating piston diesel engines crank-case lubricating oil contaminants may plate out on, or even aspirate into, combustion chambers, intermingle with fuel and fuel contaminants thus causing an increase in emission products. These contaminants may also be involved in only a partly understood phenomenon of "carbonizing". Also involved is a dynamically effective combustion in the partially known phenomenon of wall quenching, flame quenching, and fuel droplet quenching. Such contaminants may also act as condensation nuclei and become reactive within the fluid dynamics of combustion.

Automatic fuel filters and lubricating oil fuel - filters are designated as having a 5 micron size capacity, but actually cannot remove particles that are smaller than 10 - 13 microns. Fuels and lubricating oils, as they are used in engines, have contaminant residues and extraneous matter which are both larger and smaller than these sizes. The contaminants actually cause engine wear, reduced fuel economy and increased emissions. Removal of the contaminants or prevention of the formation of the particulates as they appear in fuels and oils used in diesels could have safety and economic effects on emissions, fuel economy, and engine durability.

In studying techniques of particulate removal it was discovered that oil purification equipment must remove particulate to at least the 10 micron level.

Water washing, a technique which removes particulate from fuels and oils up to 1 micron in size, is unlikely to find application aboard a motor vehicle, even though it may be useful in a large vehicle in the underground mine environment. However, hydrocarbon liquids have a relatively high dielectric value at a wide range of temperatures and permit the use of an electrostatic field to remove particles down to the 0.001 micron level.

The electrostatic fluid processing system (EFP) uses electrical charging fields, principles of dielectrics, electrostatics and electromagnetics, similar to those used in electrostatic gaseous precipitators.

A summary of railroad diesel test results showed one of the major differences in operation was the disappearance of most of the black smoke.

At the termination of the test, examination of the cathodes revealed that the EFP units had captured and retained various contaminants ranging in size from the smallest visible under the microscope to sizes in excess of 100 microns. While no calculation of the percentage removal was available, there were 10 collecting plates of approximately 16 inches diameter which contained a coating in excess of 1/8th inch thickness of contaminants removed from the fuel.

In tests on the Mexican Railroad, it was known that the high values of NO_X occurred under conditions of high smoke formation, resulting from high temperatures, heat transfer due to carbon formation and proximity to a cold surface (wall quenching). While NO_X emissions from EFP fitted diesels were not measured, it is likely that the EFP reduction in smoke formation is accompanied by some reduction in NO_X emissions.

EFP-fitted diesels emitted fewer large particles and prevented much of the submicronic particulate matter from entering the combustion chambers or appearing in exhaust emissions. The improvement occurred in diesels using a centrifuged fuel of relative good quality. Carbonation particles in sizes 10^{-9} meter and larger, including those containing high molecular weight ring compounds, are excluded from the system. Particles form instantaneously during diesel combustion and no matter how pure the fuel and lubrication oils used, no claim can be made for the EFP system preventing all of this happening.

The authors suggested that there is a possibility of a decline in fuel quality and thus new ways to purify fuels in the engine may have to be found and studies which reflect on the way fuels become contaminated are needed.

Frisch, Johnson and Leddy (47) evaluated the effects of fuels with widely-varying gravity, volatility, aromatic and sulphur contents and dilution ratios on diesel particulate emissions from a Caterpillar 3208 direct-injection, naturally-aspirated engine. Fuel 1 had a 51% (w/w) paraffin content, whereas fuel 2 contained 40% and fuel 3, 27%. The aromatic hydrocarbon content of the fuels were 15, 21 and 27%, respectively. The engine was operated at modes 3 and 5 of the EPA 13-mode cycle. The total particulate emissions increased as the volume dilution ratio increased from undiluted to diluted. Mode 5 generally showed less effect of dilution on particulate emissions than Mode 3. Fuels with a higher distillation temperature, lower API gravity and higher aromatic content tended to have higher levels of particulate matter emissions during both Mode 3 and Mode 5.

The dilution ratio had a significant effect on the measured sulphate emissions. The sulphate level decreased at Mode 3 with increasing dilution ratio, to 25:1 at Mode 5 and then decreased. The sulphur content of the fuels was 0.11, 0.27 and 0.44% by weight for fuels 1, 2 and 3, respectively. The conversion of sulphur to sulphate was relatively constant and as fuel sulphur increased, sulphate emissions increased. Also, as fuel consumption increased from Mode 3 to Mode 5, the sulphate levels increased correspondingly. With all three fuels, sulphate emissions were an important portion of the total particulate emissions, varying from 2 - 4% at Mode 3, to 9 - 12% at Mode 5.. At these modes, soluble organic fraction levels were decreased significantly, and therefore the importance of the sulphate levels increased in importance relative to their contribution to the total particulate emissions.

The engine operating mode had a significant effect on the soluble organic fraction emissions level. As the brake mean effective pressure level was increased at constant speed, both the actual level of soluble organic fractions (SOF) and the SOF emissions, as a percentage of total particulate emissions, decreased. Fuel properties also had a significant effect on the soluble organic fraction levels. Higher fuel distillation temperature, lower fuel API gravity and higher aromatic content resulted in higher emission levels.

Paraffinic hydrocarbon species accounted for about 50% by mass of the soluble organic fraction particulate emissions for all of the fuels tested at both modes. Aromatic hydrocarbon species accounted for less than 10% by mass of the SOF particulate emissions from all of the fuels tested at both modes (47).

3.3.5 Duty Cycles

The 13-mode cycle (Table 11) used by the U.S. Environmental Protection Agency (EPA) to evaluate truck engine emissions is used for, but not particularly applicable to, the assessment of diesel engines used in underground mines.

Johnson (38) provided typical power curves (Figure 29) and related them to the 13-mode cycle. The reader should be aware than in discussions concerning duty cycles, the terms torque and load are synonymous.

TABLE 11

13-Mode Diesel Emissions Cycle

Mode	Engine Speed	Percent Load[1]
1	Idle	0
2	Intermediate[2]	2
3	Intermediate	25
4	Intermediate	50
5	Intermediate	75
6	Intermediate	100
7	Idle	0
8	Rated	100
9	Rated	75
10	Rated	50
11	Rated	25
12	Rated	2
13	Idle	0

[1] Percent of maximum torque available at indicated speed.

[2] Peak torque or 60% of rated speed, whichever is higher.

(Source: Reference 38)

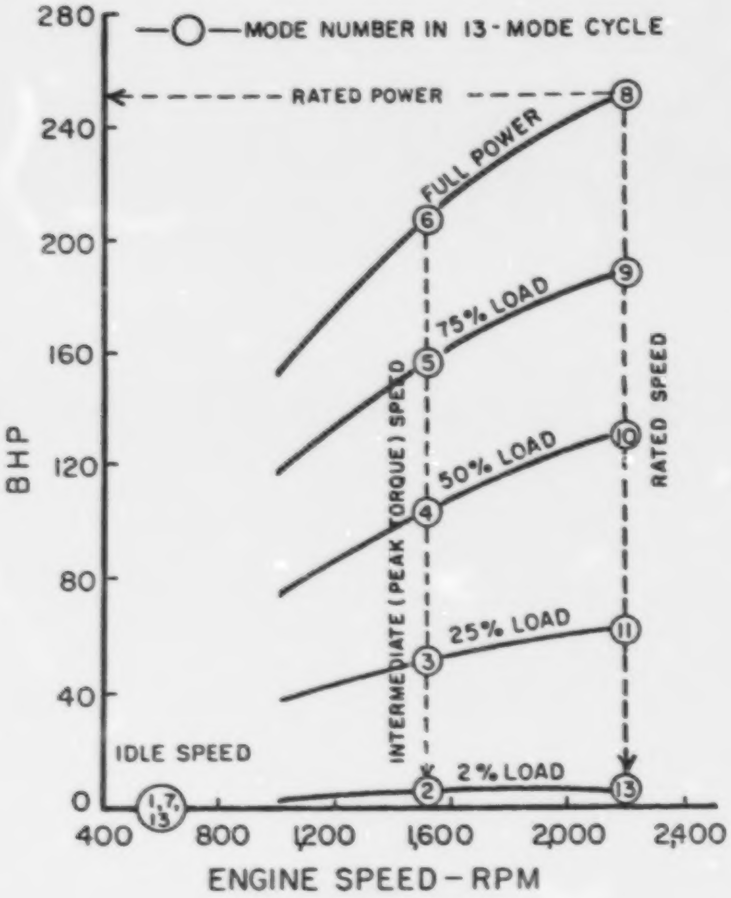


FIGURE 29

Typical diesel full- and part-load power curves

(Source: Reference 38)

CANMET personnel, in cooperation with INCO mines and the Deutz Engine manufacturer, examined the problem of duty cycles and proposed the following as being typical of an LHD use-cycle for a 700-foot haul (48) at a speed of 565 feet/minute (6 mph):

Phase 1	(preparing to load bucket) - 2000 rpm (max. torque)	10 sec.
Phase 2	(loading bucket) - 1625 rpm (max. torque)	30 sec.
Phase 3	(hauling loaded bucket, engine leading) - 2300 rpm (75% torque)	80 sec.
Phase 4	(preparing to dump bucket) - 2200 rpm (max. torque)	5 sec.
Phase 5	(dumping and shaking bucket) - 1500 rpm (50% torque)	5 sec.
Phase 6	(returning empty, bucket leading) - 2300 rpm (85% torque)	80 sec.
	TOTAL	210 sec.

For any particular distance, the only values which would change would be for Phases 3 and 6. For example, for a 200-foot haul, Phases 3 and 6 would each be carried out in approximately 25 seconds.

The emissions work group at the 1977 - NIOSH Diesel Meeting agreed that the duty cycle proposed by the CANMET group was representative of the LHD-mining situation. They suggested, however, that load factor variations would occur during various segments of the cycle, depending on the roadway structure and grade, as well as type of load (coal, ore, etc.).

To maintain continuity, and to utilize the available data base derived using the EPA 13-mode cycle, the 1977 NIOSH Workshop recommended using segments of the EPA system. It was suggested that modes 3, 6, 9, 10, plus one idle mode could be used to represent diesel mining equipment conditions. Thus, they proposed the following duty cycle characteristics:

Mode 3: 1600 rpm (25% load)
Mode 6: 1600 rpm (100% power)
Mode 9: 2200 rpm (rated speed) (75% load)
Mode 10: 2200 rpm (50% load)
Idle: 600 rpm (0% load)

Keith Alcock of Jeffrey Mining Machinery, in cooperation with the American Mining Congress Diesel Committee, released a document on duty cycles in diesel-powered vehicles in underground mines (49). The authors concluded that an engine "will operate for about 95% of its operating time at easily predetermined fixed modes."

The following modification of the 13-mode test cycle was recommended:

Mode 1: 100% load; 100% speed
Mode 2: 75% load; 100% speed
Mode 3: 50% load; 100% speed
Mode 4: 25% load; 100% speed
Mode 5: No load; high idle
Mode 6: 100% load; 90% speed
Mode 7: 100% load; 80% speed
Mode 6: No load; idle speed

A production cycle for an LDH vehicle (145 hp) over a level 600-foot haul distance was suggested as being:

	Speed	HP	Time	HP Secs.
Load		25	90	2250
Tram to changeout	6.7	145	10	1450
Tram 500 ft	6.7	145	50	7250
Turn and reverse		72	10	720
Dump		20	20	400
Tram 500 ft	8	105	42.5	4475
Turn		57	10	570
Wait		--	--	--
Tram from changeout		105	8.5	895

Total Cycle Time: 241 seconds

This can be compared with the CANMET duty cycle (48) of 210 seconds for a 700-foot haul. There are obvious differences in these two estimates of duty cycles. One example is the load time of 30 seconds at maximum torque suggested by CANMET compared to 90 seconds at 17% (25 HP) of available HP (145 HP). It should be emphasized that the Alcock report (49) did not appear to be based on field measurements, but was a tabulation based on estimates.

3.3.6 Engine Maintenance

This area is one of concern and additional research is required to better define the effects of engine wear on emission composition. At the 1981 EPA Diesel Emission Symposium, Zweidinger (50) raised the point that most of the studies published to date, in which diesel particulate was evaluated for mutagenicity in the Ames test, were from vehicles operating according to manufacturer specifications. The real-world situation is, of course, that many of the vehicles on the highway are operating under various malfunction conditions due to a lack of maintenance, component failures and engine wear and it is important to determine the effects of these malfunctions on emissions. Although little data is available detailing the effect of on mutagenicity of the tail-pipe emissions, two studies suggest that the effects could be significant. For example, the replacement of a faulty injector in a 1980 VW Rabbit Diesel resulted in a decrease of 42% in mutagenic activity of the diesel particulate. The Nissan Motor Company also reported that secondary fuel injection problems as a result of a lack of pressure pulse dampening in the fuel injection system increased the mutagenicity and BaP concentration of exhaust emissions.

There is an abundant literature supporting the need to adequately maintain underground diesel equipment. Proper maintenance not only assures the lowest emission characteristics from an engine, but also the most economic operation of the engine in terms of down-time, fuel consumption and engine life.

Comprehensive check lists for use in a diesel maintenance program were provided by Hews and Rutherford (51) and Alcock (52), and readers interested in details are referred to these publications.

The 1977 - NIOSH Diesel Emissions Workshop group suggested subdividing maintenance requirements into two main categories.

1. Preventative
2. Major overhaul

Significant to preventative maintenance were fuel and air filters, exhaust treatment systems, cooling systems, timing, injector and flame-trap servicing.

In discussing major overhauls, the group cautioned that there is evidence that rebuilt or overhauled engines do not always have emission characteristics the same as for the original equipment. Appropriate emission testing would be in order before returning overhauled engines to regular underground service.

3.4 Exhaust Emissions

3.4.1 General Considerations

It is intended that this section will provide an overview of the qualitative and quantitative aspects of the various pollutants that are emitted in diesel exhausts, and not a comprehensive discussion of diesel exhaust emissions.

As was reviewed in detail earlier, in the burning of one pound of diesel fuel, about 200 cubic feet of exhaust is produced. This is composed of about 20 cu ft of CO_2 and approximately 0.33 cu ft each of CO and NO_X. The remainder of the exhaust is made up of nitrogen (73%) and water (13%). The diesel engine is about 30 - 40% efficient in terms of the useful energy derived from diesel fuel. The remaining energy is lost with the exhaust gases and to cooling the engine air or water (38, 53).

A partial list of "typical" diesel emissions levels as given by two authorities (38, 54) can be found in Table 12.

It is obvious from the data shown in Table 12 that these two authors had differing estimates of the quantitative aspects of exhaust constituents.

In addition to the components in Table 12, an additional list of exhaust constituents of concern include: particulate matter, polynuclear aromatic hydrocarbons (PNA's), sulphur dioxide, phenols and other aromatics.

An appreciation of the effect of engine operating speed and load on the emission of various exhaust constituents can be obtained by reviewing the following series of diagrams. Although the quantitative aspects of the data shown may not be "typical" of engines used in LHD equipment, the changes in emission levels with changing operating modes are relevant.

TABLE 12

Typical Ranges of Diesel Exhaust Emissions

Component	Concentration: Reference (54)	Concentration: Reference (38)	Brake Specific Emission b/bhp-hr (Reference 38)
CO	200 - 300 ppm	50 - 5000 ppm	2 - 10
NO_X (as NO_2)	700 - 2550 ppm	200 - 3000 ppm	4 - 20
Hydrocarbons (c)	80 - 1140 ppm	10 - 500 ppm	0.2 - 3.0
Aldehydes	3 - 97 ppm	--	--
Smoke	2 - 20%	1 - 10 mg/ft^3	0.05 - 0.3

(Source: References 38 and 54)

Figures 30 and 31 show the relationship between diesel exhaust composition and the fuel:air ratio. Additional descriptions of these changes will be found in later discussions in this section when individual components are discussed, and so will not be elaborated upon at this time.

Figure 32 depicts the relationship between exhaust temperature and the F:A ratio. The manner in which smoke emissions increase with increasing power is depicted in Figure 33.

To the above data, particularly that given in Table 11, one can compare the mass emission estimates shown in Table 13, which were estimates provided by members of the Emissions Work Group at the 1977 -NIOSH Diesel Workshop.

The values shown in Table 13 represent estimates for diesel engines which are found in underground use.

Further details of the emission characteristics of both naturally-aspirated and turbocharged after-cooled heavy duty diesel engines can be found in a NIOSH Technical Report (453).

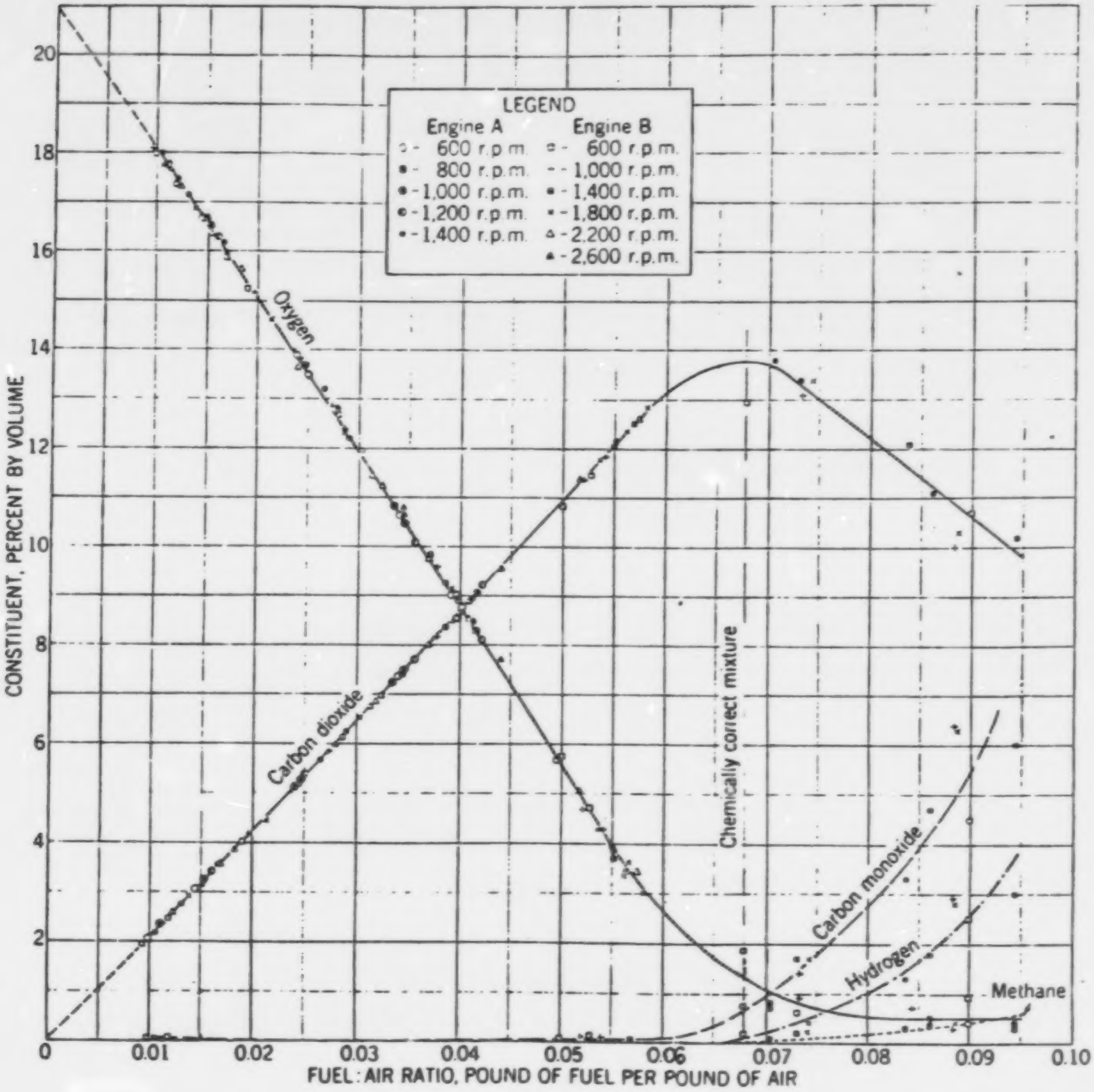


FIGURE 30

Relation of composition of diesel exhaust gas to Fuel:Air ratio

(Source: Reference 39)

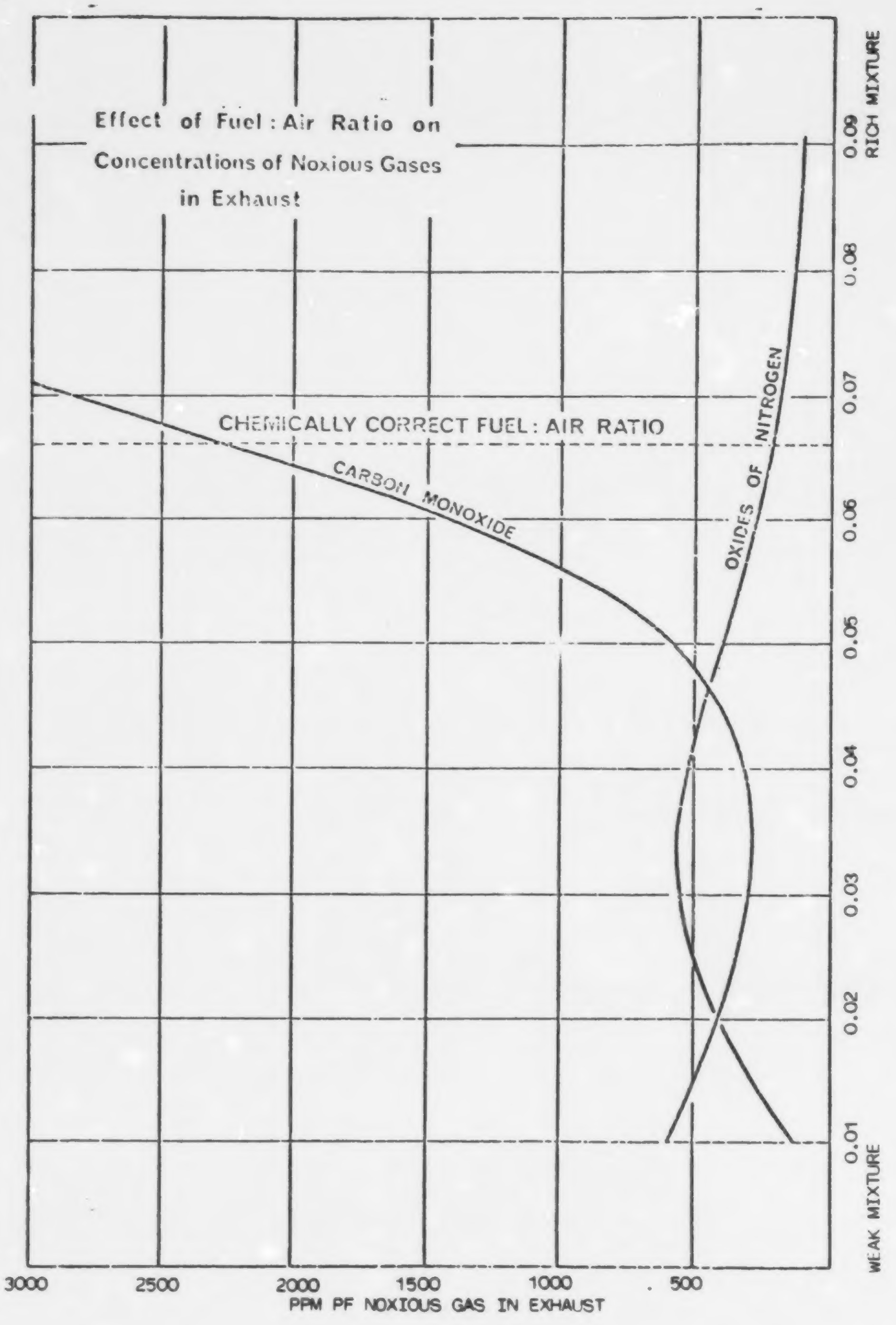


FIGURE 31

Fuel:Air Ratio - Pound of Fuel Per Pound of Air

(Source: Reference 40)

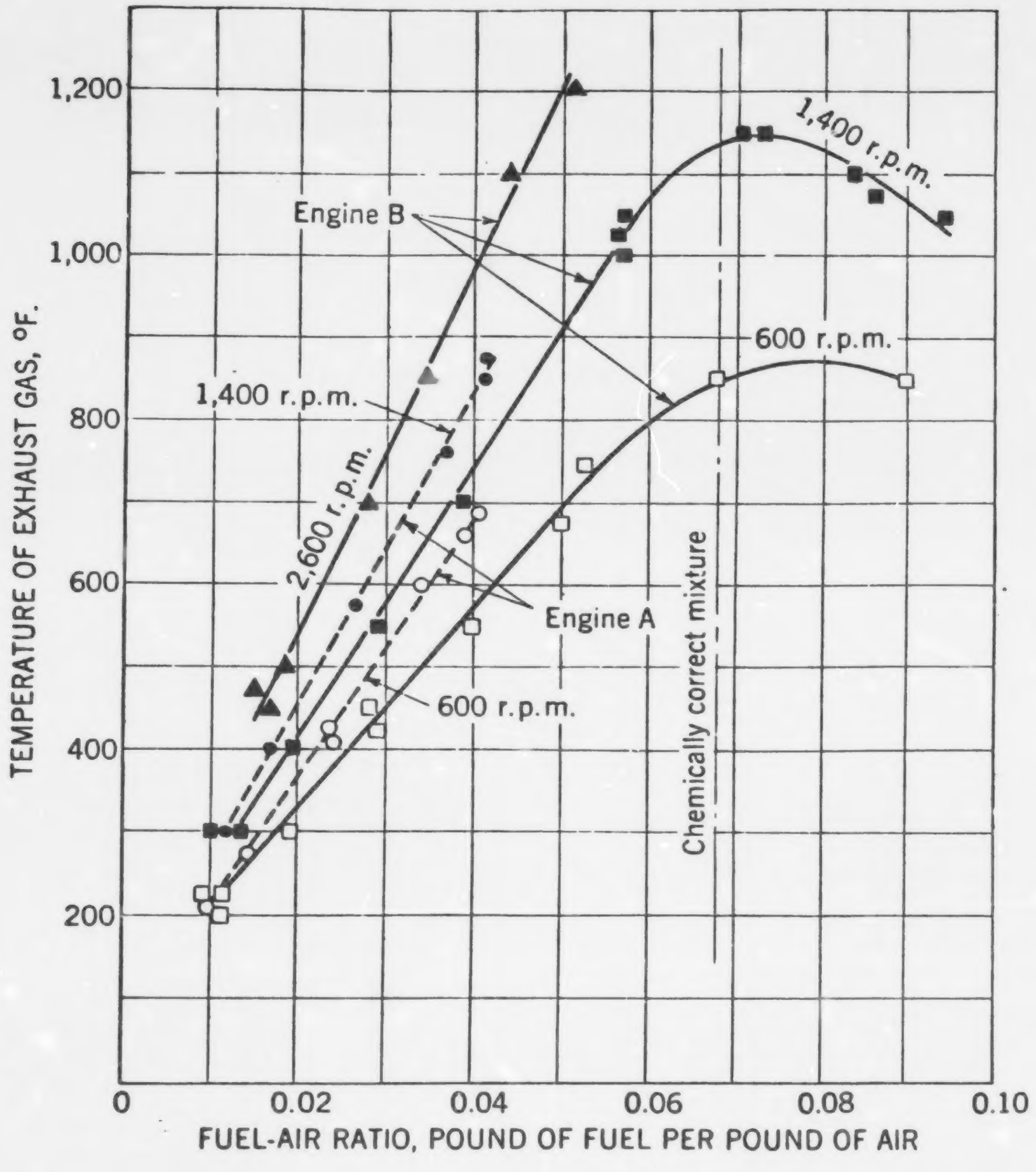


FIGURE 32

Relation of temperature of diesel exhaust gas to fuel:air ratio

(Source: Reference 39)

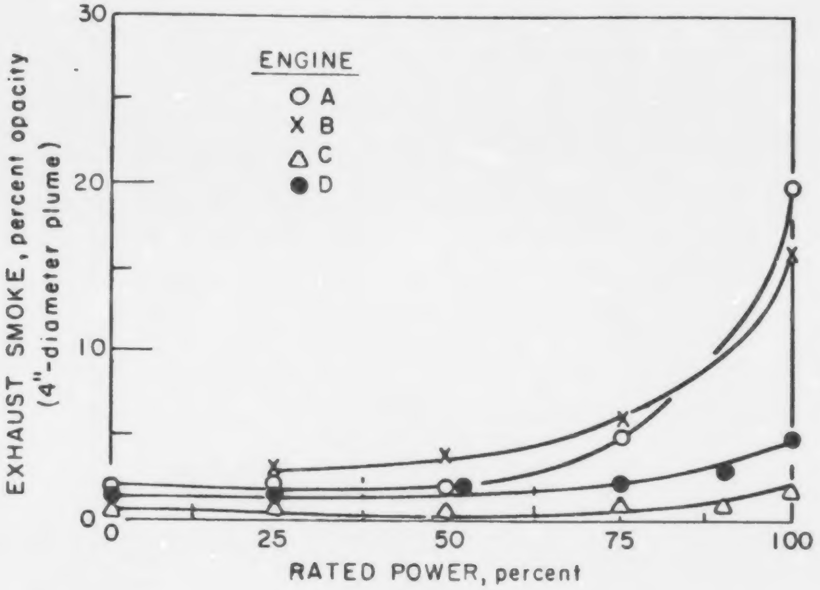


FIGURE 33

Effect of power output on exhaust smoke for 4-cycle, direct-injection, naturally-aspirated engines at rated speed

(Source: Reference 55)

Conversion of the mass emission data in Table 13 to concentrations were made by a second work group at the same NIOSH meeting and can be found in Table 14.

It will be noted that there is considerable variation in several of the values seen in Tables 12 and 13. This probably derives, in the main, from the fact that most of these values were obtained from different types of diesels, many of which are not common to mining operations.

These values may be compared to those published in a NIOSH document (453) in which the mass emissions of 9 different engines operated over modes 3, 6, 9 and 10 of the Federal EPA Heavy-duty Diesel Engine 13-Mode Cycle were given. As will be seen, most of the values compare quite well with those in Table 15, except that the NO levels are at the upper end of/or exceed those shown in Table 13. Under the column "Type of Engine", NA refers to naturally aspirated and TA to turbocharged after-cooled.

TABLE 13

Estimates of Diesel Engine Exhaust Emissions (Untreated Engine)

COMPONENT	Mass Emission (g/bhp-hr)
CO	0.6 - 2.7
CO_2	510 - 610
NO	1.25 - 4.1
NO_2	0.3 - 0.7
SO_2 (0.2% S FUEL)	0.5
PARTICULATE (80% CARBON)	0.17 - 0.67
TOTAL ALDEHYDES	0.02 - 0.2
HYDROCARBONS	0.03 - 0.17
BENZENE	1.5×10^{-4} to 1.7×10^{-3} (0.5 - 1.0% of hydrocarbons)
PHENOLS	0 - 0.1 (trace)
PNA's	Trace

(Source: 1977 - NIOSH Diesel Workshop)

TABLE 14

Estimates of Diesel Engine Ehaust Component Concentrations As Calculated from Values Given in Table 13

Component	Range in Undiluted Exhaust
CO	114 - 513 ppm
CO_2	6.1 - 7.2%
Hydrocarbons	(emission data questionable)
Benzene	(emission data questionable)
NO	225 - 735 ppm
NO_2	36 - 84 ppm
SO_X (based on 0.2% S in fuel*)	7.7 ppm
SO_X (without scrubber)	40 ppm
Particulate*	0.12 - 0.47 g/bhp-hr
Aldehydes (total)	(emission data questionable)
Formaldehyde	(emission data questionable)
H_2SO_4	(emission data not available)
Aromatics	(emission data not available)
PNA	(emission data not available)

* For engines equipped with scrubbers

NOTE: Conversion of g/bhp-hr to ppm were made using the following calculations: (Typical of de-rated 75 - 180 hp IDI engines)

All gases: 1 g/bhp-hr = 185 ppm (wt)

CO 1 g/bhp-hr = 190 ppm by vol
NO 1 g/bhp-hr = 180 ppm by vol
NO_2 1 g/bhp-hr = 120 ppm by vol
NO_X 1 g/bhp-hr = 174 ppm by vol
SO_2 1 g/bhp-hr = 80 ppm by vol
CO_2 1 g/bhp-hr = 120 ppm by vol

All figures assume exhaust gas emission of 70 l/bhp min (2.5 cfm/6 hp).

(Source: 1977 - NIOSH Diesel Workshop)

TABLE 15

Diesel Engine Exhaust Emissions*

Engine	Cylinder	Type	NO (gm/bhphr)	CO_2 (gm/bhphr)	CO (gm/bhphr)
B-4	4	NA	4.2	600	1.6
F-4	4	NA	5.8	750	1.7
F-4G1	4	NA	4.5	690	7.6
B-6	6	NA	4.2	640	1.9
A-6	6	NA	3.8	670	1.5
E-6	6	NA	5.6	812	2.4
E-6TA	6	TA	5.4	580	0.8
D-8TA1	8	TA	6.0	--	--
A-12	12	NA	4.6	700	2.3

* Based on Modes 3, 6, 9 and 10 of the Federal EPA heavy-duty diesel engine 13-mode cycle

(Source: Reference 453)

The key issue concerning diesel emissions is the concentration found in the workplace to which the miner is exposed. Ultimately, mine atmosphere measurements are required in assessing the health effects of diesel pollutants. However, a discussion of diesel-derived constituents at the "tail pipe" serves two major purposes. Firstly, it permits the study of engine operating conditions and the effect they have on the quality and quantity of the exhaust constituents. Secondly, by quantitating the exhaust contents, an estimate of the amount of dilution can be calculated, and therefore the volume of mine ventilation required, which would result in a final environmental exposure concentration for each component of concern.

The above estimates and approximations of diesel exhaust constituents can be compared to the actual exhaust concentrations obtained by Stewart, Dainty and Mogan (48), using typical underground IDI (Deutz) and DI engines. The data, shown in Tables 16 and 17, were obtained using a duty cycle considered typical of LHD operation in a mine, which was discussed in Section 3.3.5.

The integrated emissions for both engine types over two haul distances, 200 and 700 feet, are presented in Table 18. The values presented by Stewart et al compare favourably with those agreed to at the 1977 - NIOSH Diesel Workshop.

There are indications that the DI engine presently marketed by General Motors (commonly referred to as the Detroit or GM "clean" diesel) may be gaining acceptance in mine use because of economic considerations. The authors, therefore, consider it appropriate to elaborate somewhat on this particular engine to permit comparison with the Deutz-type IDI engine.

It has been suggested that, although the GM DI diesel may emit pollutants in the same general concentration ranges as IDI engines, the DI machine has a greater total or mass emission than the IDI diesel due to the large volume of exhaust expelled. According to GM, their DI engine has an exhaust volume about 1.3 times the Deutz. INCO, however, uses a figure of 1.7 times.

Table 16: IDI Engine and Emission Characteristics for the Various LHD Cycle Phases

Phase	Engine Speed (rpm)	Engine Torque (lb ft)	Engine Fuel Consumption (lb/hr)	Engine Air Consumption (lb/hr)	Exhaust Emissions on Dry Basis			
					CO (ppm)	NO (ppm)	NO_2 (ppm)	Particulates (mg/m³)
1	2000	318 (max.)	55.0	1110	330	557	0	125.7
2	1625	328 (max.)	43.7	923	235	555	0	153.9
3	2300	234	48.2	1278	196	683	0	80.0
4	2200	309 (max.)	59.8	1200	400	613	0	113.0
5	1500	171	22.9	901	80	750	0	33.0
6	2300	270	55.2	1263	210	637	0	90.0

Table 17: DI Engine and Emission Characteristics for the Various LHD Cycle Phases

Phase	Engine Speed (rpm)	Engine Torque (lb ft)	Engine Fuel Consumption (lb/hr)	Engine Air Consumption (lb/hr)	Exhaust Emissions on Dry Basis			
					CO (ppm)	NO (ppm)	NO_2 (ppm)	Particulates (mg/m³)
1	2000	318	50	1012	1400	1150	0	200
2	1625	328	36	770	800	1230	0	130
3	2300	234	44	1176	290	940	0	65
4	2200	309	56	1125	1400	1060	0	200
5	1500	171	18	720	430	780	60	40
6	2300	270	51	1176	550	1050	0	100

Table 18: The Integrated Engine & Emission Characteristics of Equal-Power-Output DI and IDI Diesel Engines for LHD Operation

Haule Distance (feet)	Engine Type	Engine Fuel Consumption (lb/hr)	Engine Air Consumption (lb/hr)	Exhaust Emissions on Dry Basis					
				CO (ppm)	NO (ppm)	NO_2 (ppm)	Particulates (mg/m³)	CO_2 (%)*	SO_2 (ppm)*
200	IDI	48.6	1130	229	620	0	111.6	9.0	98
200	DI	43.2	1013	681	1073	3	112.0	8.9	97
700	IDI	50.1	1203	215	641	0	97.7	8.7	95
700	DI	45.3	1097	545	1031	1.4	96.7	8.6	95

*Calculated from engine fuel and air consumptions for diesel fuel with 86% carbon and 0.25% sulphur. All fuel carbon and sulphur are assumed to combust to CO_2 and SO_2.

(Source: Reference 48)

The comparative literature on engine emissions has implicated the DI engine as being less "clean" than IDI diesels (56). In 1973, [illegible]n (38) noted that, at the time, emissions were lowest from prechamber, turbocharged, after-cooled engines with levels generally as follows:

$$HC = 0.2 - 0.3 \text{ g/bhp-hr}$$
$$CO = 1 - 2 \text{ g/bhp-hr}$$
$$NO_X \text{ (as } NO_2) = 5.5 - 6.0 \text{ g/bhp-hr}$$

Additionally, he stated that "some direct-injection, turbocharged, after-cooled engines appear to be able to approach similar numbers although most engines with NO_2 in the above range have not been proven in field service." Modifications that lower NO_2 output usually leads to increased fuel consumption and smoke.

During the 1977 - NIOSH Diesel Meeting, the following comparison of DI and IDI engines was provided, which indicates clearly the attitudes of those in the emissions work group.

"The following is an attempt to compare the two combustion systems without emission control systems.

Characteristic	Direct Injection	Indirect Injection
Fuel economy	Favourable	Less favourable
CO	Less favourable	Favourable
HC	Less favourable	Favourable
NO and NO_2	Less favourable	Favourable
Particulates*	---	---
Aldehydes	Less favourable	Favourable
SO_2	Approximately same	Approximately same

* To be determined with known duty cycle.

Conclusion: Based on the above comparison, it can be concluded that the indirect injection engine has advantages for underground mining usage and is likely to retain these advantages for at least the next few years. Certain pollutants are still to be determined when appropriate instrumentation and procedures are available and a suitable duty cycle is defined."

— The Detroit diesel is a 2-stroke (cycle), direct-injection (DI) engine and employs a forced-air, or air-scavenging system to assist in exhaust dilution and removal. According to the manufacturer, it has better rpm-torque (Figure 34) and acceleration (Figure 35) relationships than IDI engines.

In a study carried out by the Ontario Research Foundation (ORF) and Texasgulf (Canada) Limited (57), the emission figures shown in Table 19 for 6 and 8-cylinder series 71-Detroit diesel engines were reported. The GM engines were operated over portions of the EPA 13-mode cycle using Number 1 diesel fuel with a sulphur content of 0.30%.

In the same report, values for a 10-cylinder Deutz F10L-714 diesel engine were also given (Table 20). In addition, findings were published for three 6-cylinder Caterpillar engines. Values for the Caterpillar 3306T engine, which produced equivalent power to the 10-cylinder Deutz and V8 GM diesels, can be found in Table 21.

All data is from engines without scrubbers of any kind.

More recent reports (CANMET - personal communication) indicate that the GM diesel NO data in the Texasgulf study discussed above may be low. In a later investigation, the NO output was found to be 9.03 g/bhp-hr at 2150 rpm and 222 bhp. In the same tests, CO was 1.27 g/bhp-hr. At 2150 rpm and 166 bhp, particulate levels were 0.18 g/bhp-hr.

Hare, Springer and Bradow (58), in evaluating a turbocharged version (which normally emits less particulates than engines not equipped with a turbocharger) of the GM engine (6L-71T), found a particulate output of 59 mg/m^3, which is about 3-fold more than the values reported by ORF. In the Hare study (58), 55% of the particulate was organic solvent soluble, whereas ORF found about 90% organic solubles.

Hare et al (58), found that in tests in which no dynamomter was used, that the CO output was 2.20 and NO$_X$ 11.8 g/bhp-hr, using No. 1 fuel.

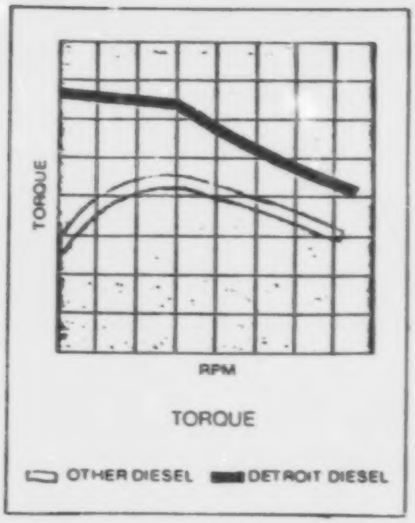


FIGURE 34

RPM - Torque relationship for IDI and GM Diesels

(Source: Reference 40)

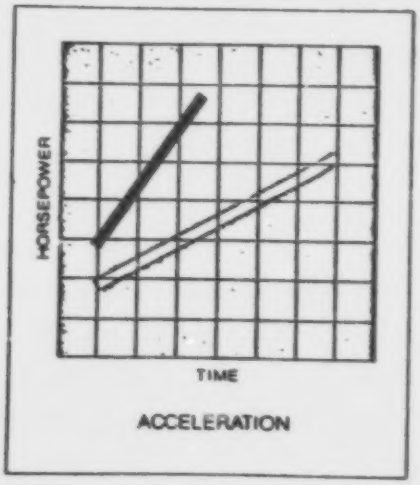


Figure 35

Acceleration - horsepower relationship for IDI and GM Diesels

(Source: Reference 40)

TABLE 19

GM - DI Diesel Emission Data

			CO_2		CO		SO_2		Total HC (as methane)		Particulates		NO		NO_X as NO_2
	bhp	rpm	%	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr	mg/m^3	g/bhp-hr	ppm	g/bhp-hr	g/bhp-hr
	180	2150	4.15	529.0	240	1.95	21	0.39	9	0.04	30.1	0.21	550	4.78	7.33
	140	2280	3.60	608.2	90	0.97	18	0.44	9	0.06	24.4	0.23	400	4.61	7.07
GM-	150	1600	4.35	508.9	565	4.21	22	0.38	130	0.57	33.5	0.21	675	5.39	8.26
V6	85	1400	2.95	522.9	52	0.59	15	0.39	72	0.47	10.7	0.10	415	5.02	7.69
	172	2000	4.35	509.8	260	1.94	22	0.38	55	0.24	25.6	0.16	590	4.72	7.23
	125	2280	3.30	606.8	68	0.80	17	0.46	60	0.41	24.7	0.25	310	3.90	5.98
	235	2130	4.50	554.9	220	1.73	23	0.41	200	0.93	28.5	0.19	550	4.63	7.09
	180	2150	3.65	561.7	98	0.96	19	0.43	190	1.09	20.1	0.17	370	3.88	5.95
GM-	205	1600	4.88	474.3	300	1.86	25	0.35	190	0.70	31.7	0.17	710	4.71	7.22
V8	108	1500	2.80	505.4	220	2.53	14	0.37	278	1.86	41.2	0.41	355	4.37	6.70
	232	1960	4.75	507.4	270	1.84	24	0.37	145	0.58	34.3	0.20	615	4.48	6.87
	186	2250	3.70	548.5	102	0.96	18	0.39	195	1.08	25.8	0.21	350	3.54	5.43

(Source: Reference 60)

TABLE 20 Deutz F10L-714 Emission Data

Test No.	bhp	rpm	CO$_2$		CO		SO$_2$		Total HC (as methane)	
			%	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr
1	203	2040	9.03	608.4	300	1.29	47	0.46	230	0.60
2	150	2090	6.00	640.2	158	1.07	31	0.48	130	0.53
3*	183	1720	7.40	589.1	140	0.71	37	0.43	51	0.16
4	103	1500	4.25	597.9	96	0.86	24	0.45	80	0.42

Test No.	bhp	rpm	Particulates		Nitric Oxide		Oxides of Nitrogen (NO + NO$_2$)		Oxides of Nitrogen (expressed as NO$_2$)
			%	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr	g/bhp-hr
1	203	2040	97.2	0.36	510	2.34	510	2.34	3.59
2	150	2090	41.0	0.24	450	3.27	450	3.27	5.02
3*	183	1720	87.2	0.38	340	1.85	340	1.85	2.83
4	102	1500	43.3	0.33	350	3.36	350	3.36	5.15

(*) - Aldehydes on Test 3; 5 ppm as formaldehyde; 0.03 g/bhp-hr

(Source: Reference 57)

TABLE 21

Caterpillar 3306T Emission Data

Test No.	bhp	rpm	CO$_2$		CO		SO$_2$		Total HC (as methane)	
			%	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr
117	255	2200	7.00	572.6	64	0.33	35	0.43	29	0.09
18	230	2235	6.55	541.4	66	0.35	33	0.40	20	0.06
19*	230	1700	7.40	493.6	56	0.24	38	0.37	14	0.04
20	110	1560	5.70	530.4	66	0.37	28	0.36	28	0.09
21	250	2000	7.10	514.3	54	0.25	36	0.38	17	0.05
22	200	2220	6.10	549.0	80	0.46	31	0.41	20	0.07

Test No.	bhp	rpm	Particulates		Nitric Oxide		Oxides of Nitrogen (NO + NO$_2$)		Oxides of Nitrogen (expressed as NO$_2$)
			%	g/bhp-hr	ppm	g/bhp-hr	ppm	g/bhp-hr	g/bhp-hr
17	255	2200	19.5	0.09	760	4.24	760	4.24	6.50
18	230	2235	18.2	0.08	725	4.09	725	4.09	6.27
19*	230	1700	21.4	0.08	675	3.07	675	3.07	4.71
20	110	1560	20.0	0.10	555	3.34	555	3.34	5.13
21	250	2000	23.8	0.09	755	3.73	755	3.73	5.72
22	200	2220	29.2	0.14	660	4.05	660	4.05	6.21

(*) - Aldehydes on Test 19; 3 ppm as formaldehyde; 0.01 g/bhp-hr

(Source: Reference 57)

In work published by Springer and Stahman (124), a GM 6V71-N diesel was found to emit 81.74 mg/m^3 particulate at intermediate speed and 50% load and 143.7 mg/m^3 at high speed and 50% load. The average NO_2 emission (using the 13-mode EPA duty cycle) was 15.8 g/bhp-hr. Both these emissions are considerably higher than those reported in the Texasgulf study (57).

In view of the discrepancies and differences in the reported emissions from GM-DI diesel engines, it would be prudent to have integrated emission levels measured in the underground environment for these engines before they are given widespread acceptance.

Recalculation of the Stewart et al (48) data (found in Tables 16, 17 and 18), in mass emission terms (g/bhp-hr) is given in Tables 22, 23 and 24 to permit those interested to more readily compare their data with other publications (Mogan - personal communication), but it will not receive further discussion in this report.

As an added point of interest, emission data published by Marshall and Fleming (55) in 1971, is given in Table 25 for comparison with the more recent values provided above.

TABLE 22

IDI Engine Mass-Emissions for LHD Cycle Phases

(see Table 16 for concentrations)

			Exhaust Emissions		
Phase	bhp*	rpm	CO g/bhp-hr	NO g/bhp-hr	Particulate g/bhp-hr
1	121	2000	1.4	2.5	0.46
2	101	1625	1.0	2.5	0.60
3	102	2300	1.1	4.2	0.39
4	129	2200	1.7	2.8	0.42
5	49	1500	1.7	6.7	0.23
6	118	2300	1.1	3.4	0.38

$$*\text{bhp} = \frac{\text{torque x rpm}}{5252}$$

TABLE 23

DI Engine Mass-Emissions for LHD Cycle Phases

(see Table 17 for concentrations)

Phase	bhp*	rpm	Exhaust Emissions CO g/bhp-hr	NO g/bhp-hr	Particulate g/bhp-hr
1	121	2000	5.4	4.8	0.66
2	101	1625	2.8	4.6	0.39
3	102	2300	1.5	5.3	0.29
4	129	2200	5.7	4.7	0.69
5	49	1500	2.9	5.6	0.23
6	118	2300	2.5	5.2	0.39

$$^{*}\text{bhp} = \frac{\text{torque} \times \text{rpm}}{5252}$$

TABLE 24

Integrated Mass-Emissions for IDI and DI Engines

(see Table 18 for concentrations)

Haul Distance (feet)	Engine Type	Exhaust Emissions CO g/bhp-hr	NO g/bhp-hr	NO_2 g/bhp-hr	Particulate g/bhp-hr
200	IDI	1.14	3.36	0	0.46
200	DI	2.81	5.00	0.40	0.03
700	IDI	1.11	3.58	0.42	0
700	DI	2.67	5.34	0.37	0.02

TABLE 25

Brake Specific Emission Rates for Diesel Engines

Engine	Gaseous emissions, g/bhp-hr (18)					Smoke emissions (18)	
	Hydrocarbons	Carbon monoxide	Nitric oxide (as NO_2)	Particulates	Carbon dioxide	Acceleration mode, percent opacity	Lug mode, percent opacity
4-STROKE CYCLE, NATURALLY ASPIRATED, DIRECT-INJECTION							
A.....	1.9	10.0	8.7	0.6	755	16.0	18.5
B.....	3.1	4.2	6.7	.6	595	10.0	12.5
C.....	3.0	5.8	19.8	.1	633	3.0	3.0
D.....	3.1	4.9	16.7	-	633	3.0	3.0
E.....	2.3	5.6	9.5	.2	570	25.0	12.4
F.....	.6	5.7	6.9	-	693	11.4	13.3
4-STROKE CYCLE, TURBOCHARGED, DIRECT-INJECTION							
G.....	3.1	3.9	17.8	.2	691	14.7	5.5
H.....	2.6	4.9	11.7	.3	677	14.7	5.5
4-STROKE CYCLE, TURBOCHARGED, PRECOMBUSTION CHAMBER							
I.....	0.3	2.3	6.1	0.4	694	19.7	2.2
2-STROKE CYCLE, DIRECT-INJECTION							
J.....	0.7	6.1	14.7	-	640	8.8	3.0

(Source: Reference 55)

The literature on exhaust emissions is extensive. However, it is doubtful if any further detail than has already been presented would serve any useful purpose. In fact, because the literature includes data from such a wide variety of engine types, many of which are not typical to underground applications or are used in vehicles which are not primary contributors to mine pollutant concentrations, to list additional tables of values would only add confusion to an area already difficult to interpret.

Much has been written on the effect of engine design on performance, efficiency and emission levels. It would be presumptive for the present authors to make technical judgements in an area requiring such specialized knowledge.

Engine manufacturers and engineers obviously are cognizant of the effects of engine design on emission characteristics. The major problem appears to be that, in many instances, a change which results in a reduction in one exhaust component leads to an increase in a second constituent, or to an unacceptable change in performance or fuel economy. Every engine modification requires a knowledgeable assessment of the benefits to deleterious effects resulting from any particular design change.

Authoritative comments, such as were received from Mr. R. W. Wheeler of Ricardo Engineering (England) (61) are significant. In his opinion a 50% reduction in particulates and NO_X is feasible, but probably with a fuel "penalty". He also expressed a need for lower sulphur fuels in order to attain cleaner exhausts, particularly if catalytic converters are used.

The Emissions Work Group at the 1977 - NIOSH Diesel Meeting, in discussing engine emission improvements, concluded:

> "There are many options available which have high potential for considerably improving diesel engine exhaust emissions. These fall under three headings:
>
> > Improvements related to the engine intake system;
> > In-cylinder control of the combustion process;
> > Post-cylinder exhaust emission control.
>
> In the short term, the greatest potential appears to lie in post-cylinder exhaust emission control."

They further recommended:

> "that research should immediately proceed in the area of developing a combined emission control system comprising advanced catalyst, water scrubber, and particulate filtration technology. Some potential also lies in the use of water/fuel emulsification and water injection techniques together with EGR for reducing NO_X emissions.
>
> In the long term, development of alternate low emission engines may prove to be more effective than employment of complex retrofit emission control devices. However, the market for such engines is small, and may be unattractive to major engine manufacturers. Employment of retrofit devices and engine derating therefore appears to be the only solution to minimizing underground diesel emissions in the next decade.
>
> Further, emission improvement that can be accomplished by such control of diesel engines may result from the development and use of other prime movers such as flame-shielded methanol and external combustion engines."

It was also stated that:

> "current emission control technology is available to substantially reduce recognized pollutant compounds in a diesel exhaust gas stream using either singular devices or a combination of devices and methods by catalytic oxidation, water application, and engine control with a resultant decrease in exhaust gas temperature at the tailpipe."

In response to this statement, it was recommended that:

> "further research should be concentrated on the development of the combination of devices and for engine controls, water jacketed for coal mine application. Emphasis is necessary on the reduction of NO_X ($NO + NO_2$) and particulate matter. Research is needed on the analysis and reduction of PNA's and phenols. Sulphur emissions to be controlled at source by drastic reduction in sulphur in diesel fuel."

The interested reader is referred to the published literature for more details. Reference 38 provides a basic insight into the influence of engine design and emissions, as well as giving additional relevant references.

It is worthwhile to peruse Figure 36 which relates exhaust component emissions with their relative toxicity (62). The toxicity of each exhaust constituent was defined as the ratio of raw exhaust concentration for each constituent to its concentration at the maximum safe permissible limit. The following ACGIH - TLV's were chosen for this latter limit:

CO	5000 ppm
CO	50 ppm
Particulates (carbon black)	3.5 mg/m^3
NO	25 ppm
NO_2	5 ppm

For comparison purposes, it was assumed that the toxicity of each component increased linearly with the number of TLV's found in the raw, undiluted exhaust.

As seen in Figure 36, the particulate fraction was the largest single toxic component over the entire F:A ratio range.

Perez and and Bower (63) provided a summary of the affect of various engine control changes on emisson characteristics (Table 26).

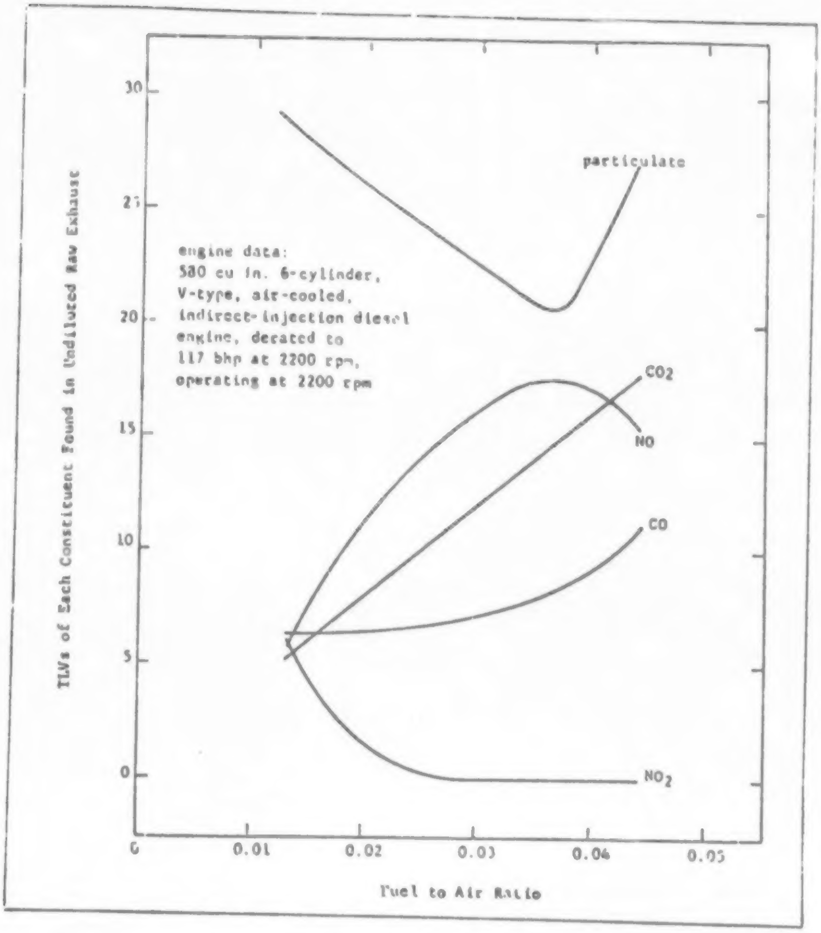


Figure 36

The relative toxicity of the various constituents found in the exhaust from an indirect-injection diesel engine

(Source: Reference 62)

TABLE 26

Engine Change Tradeoffs

↑ = Increase ↓ = Decrease ↔ = No Change

Change	Particulates Total	Particulates SEF	HC	NO_x	ALD	BAP	Fuel Cons	Power
PG-DI	↑	↑↓	↑↓	↑	↑↓	↑	↓	↔
EGR	↑	↓	↓	↓	↓	↓	↑	←↓
Timing								
Advance	↑↓	—↑	↔	↑	↑↓	↑	←↑	↔
Retard	↑↓	↑	↑↓	↓	—↑	↑	↑	↓
Aftercooling	↑↓	↑↓	↑↓	↑↓	↓	↓	↑↓	↑
Injector SAC Volume Increase	↑	↑	↑	↑	↑	↑	↔	↔
Catalyst*	↑↓	↑↓	↓	↓	↓	↓	←↑	↔
Fuel (BaP Increase)	—	—	—	—	—	↑	—	—

* SO_4 ↑↓ , NH_3 ↑

(Source: Reference 63)

3.4.2 Carbon Dioxide

As was illustrated in Figure 36, CO_2 concentrations increase with increasing F:A ratio. Concomitantly, a decrease in O_2 levels also occurs. Carbon dioxide is one of the end products of the perfect combustion of diesel fuel and its concentration in exhaust gas can be readily estimated by assuming that all of the carbon in the fuel is converted to CO_2.

For a diesel fuel with 85.5% by mass carbon and 14% by mass hydrogen, the combustion of 1 g of fuel would yield 3.135 g CO_2 and

1.26 g H_2O (64). With the usual underground derated diesel with a F:A ratio of 0.045, the CO_2 would be diluted to produce an exhaust concentration of about 8.9% CO_2 (by volume) and 6.8% O_2 (by volume).

The only way in which CO_2 emissions can be reduced is by reducing the total diesel fuel consumption or changing the carbon content of the fuel.

It has been suggested by Hurn (53) that the CO_2 levels in mine atmospheres, which is easily and reliably measured, and is two orders of magnitude greater than CO, may be a good indicator of pollution. This point will be discussed further in the section on the pollutant levels in mine atmospheres.

The relative relationships between increasing CO_2 and decreasing O_2 levels and CO and NO_X are shown in Figures 37 and 38.

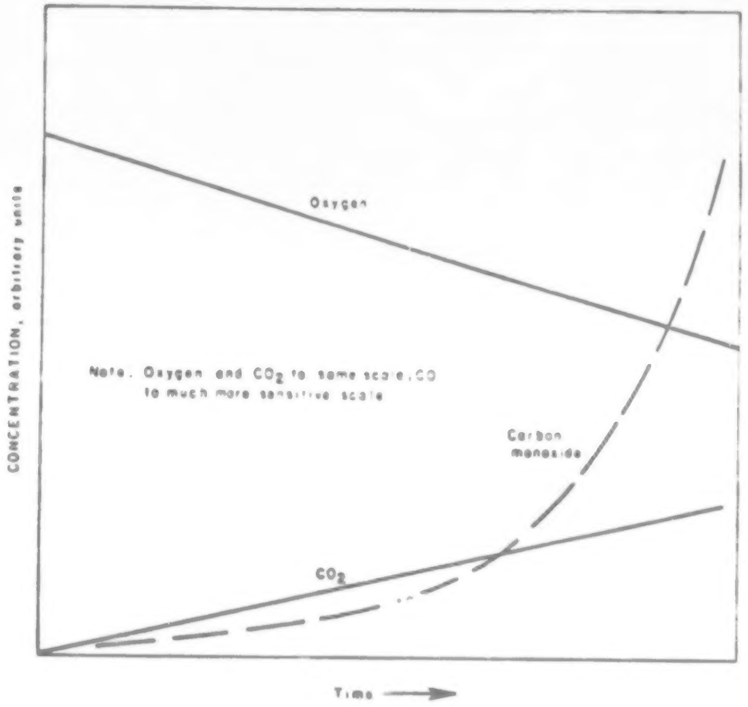


FIGURE 37

Contamination and oxygen depletion accompanying diesel engine operation in closed system (carbon monoxide buildup)

(Source: Reference 53)

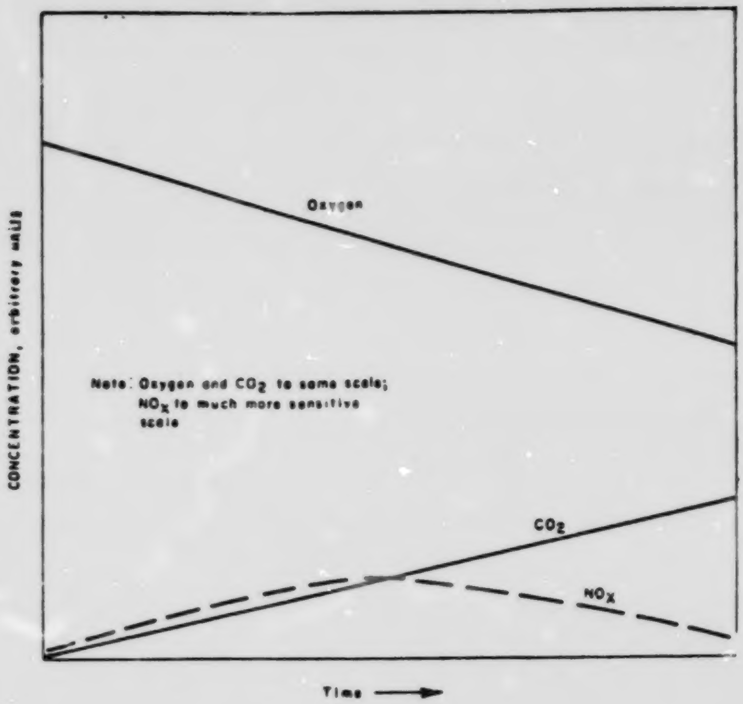


FIGURE 38

Contamination and oxygen depletion accompanying diesel engine operation in closed system (trend for nitrogen oxide)

(Source: Reference 53)

3.4.3 Carbon Monoxide

As seen in Figure 39, carbon monoxide emissions increase with increasing F:A ratio and become particularly pronounced with rich fuel mixtures as a result of incomplete combustion.

The relationship between CO formation with fuel consumption and oxygen depletion can be viewed in Figure 40.

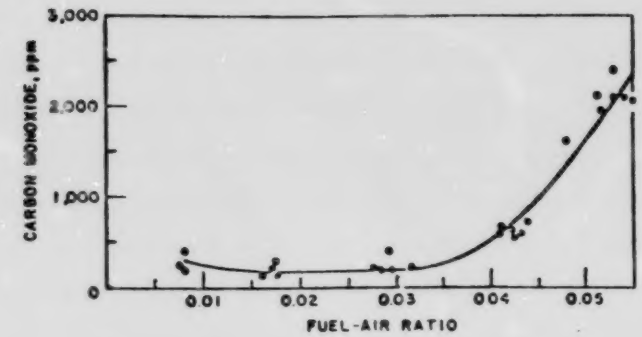


FIGURE 39

Dependence of carbon monoxide emission on F:A ratio

(Source: Reference 65)

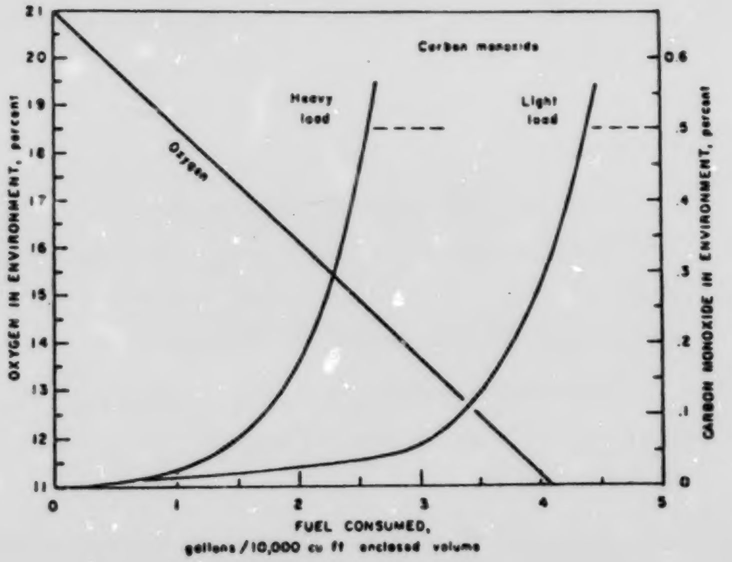


FIGURE 40

Development of hazardous CO concentrations by an engine operated in closed environment

(Source: Reference 56)

Of more quantitative relevance to the mine situation is the data of Mogan et al (56), in which the CO emission from a derated IDI engine equipped with a catalytic converter was measured. Figure 41 shows how the CO content of the exhaust rises rapidly at both high and low speeds as the F:A ratio approaches the chemically-correct value. This explains the need to derate diesel engines for underground use.

Figures 42 and 43 provide CO emission data versus fuel:air ratios presented in mass emission g-bhp/hr and g/hr for comparative purposes for a 6-cyclinder NA-IDI engine (E-6), a 6-cylinder TA-IDI engine (E-6TA) and a 4-cylinder NA-IDI engine (B-4) (453).

It should be recalled that, as the speed of the engine increases, the total CO emitted also increases (Figure 44). Mogan and co-workers (56) pointed out that "even at high speed, high load, derated machines rarely produce enough CO to govern ventilation requirements."

In describing the INCO operation, Hews and Rutherford (51) reported that from 4,500 tests for CO in undiluted engine exhaust taken in 1972, they found that for LHD equipment, 96% of the readings were below 300 ppm. For locomotives, 94% were less than 800 ppm and 98% were below 1,000 ppm for drills and tractors, etc. (In Ontario, the maximum permitted CO content of undiluted exhaust is 1500 ppm).

Marshall (65) measured CO output from a 4-stroke cycle (air-coooled) IDI engine and found a relationship with power as shown in Figure 45. A DI, 2-stroke cycle engine had CO emissions with increasing power as shown in Figure 37.

The effect of a platinum catalytic converter in lowering the CO output for both the DI and IDI engines can be evaluated by perusing Figures 45 and 46.

Figure 47 illustrates CO emission changes as a result of speed and load in examples of naturally-aspirated and turbocharged after-cooled afterburner engines. Additional details of the CO emissions of a naturally-aspirated engine versus speed and load can be reviewed in Figure 48.

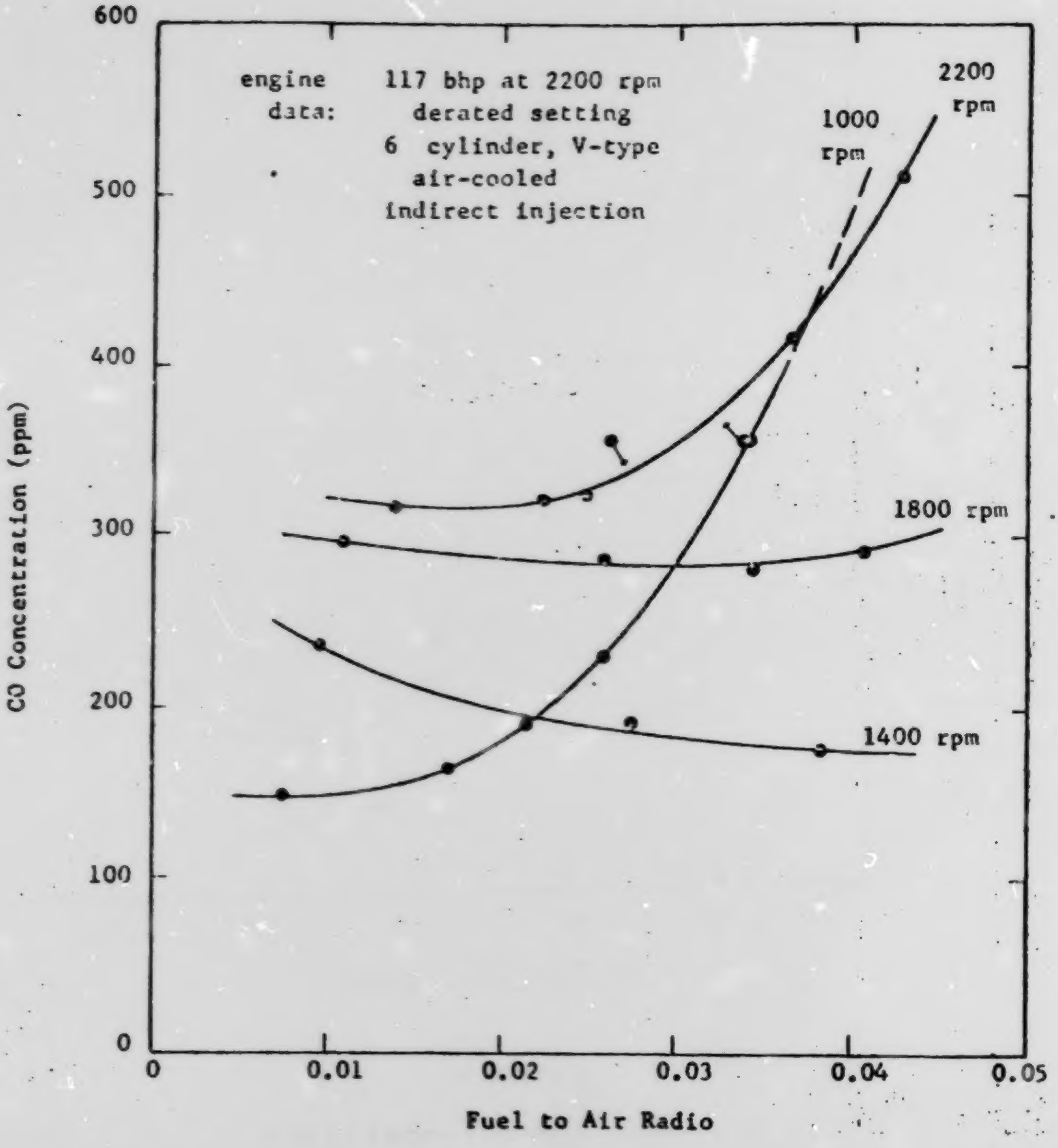


FIGURE 41

Variation of engine exhaust CO concentration with F:A ratio and speed

(Source: Reference 56)

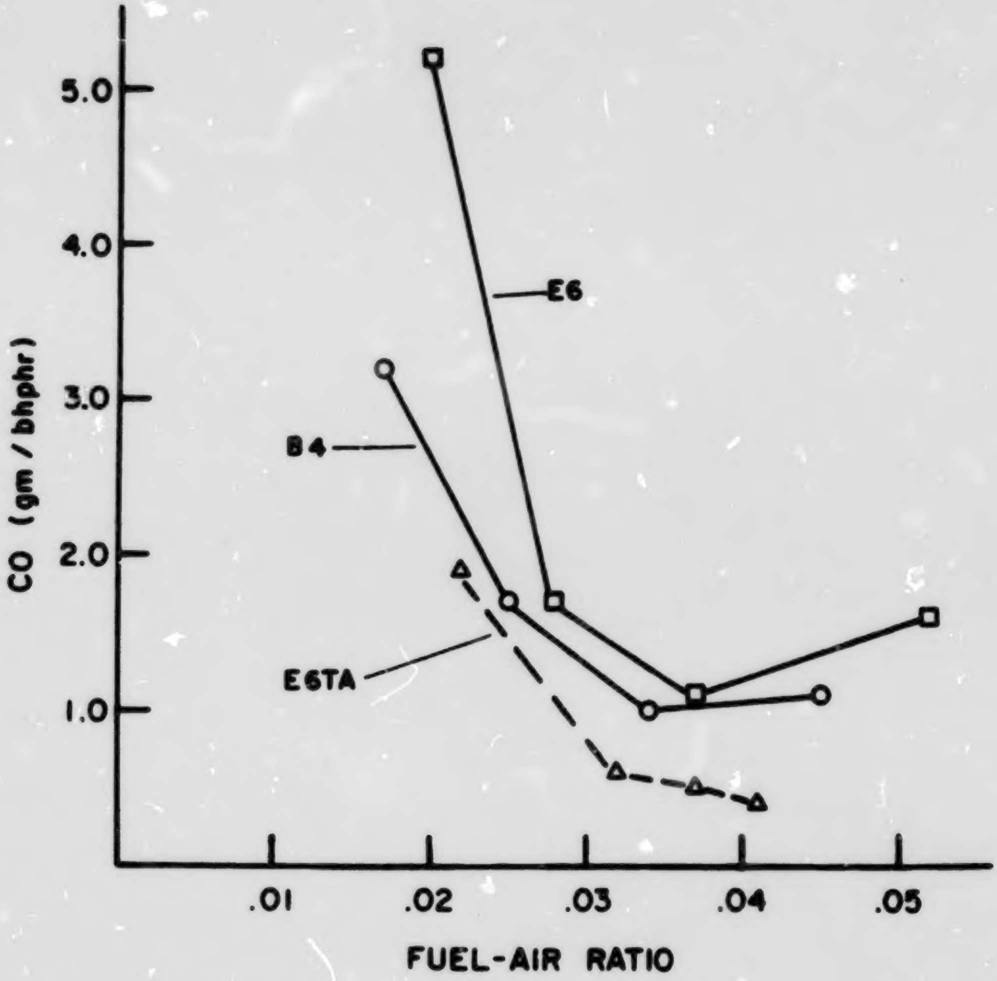


FIGURE 42

CO emissions (gm/bhphr) versus F/A ratio

(Source: Reference 453)

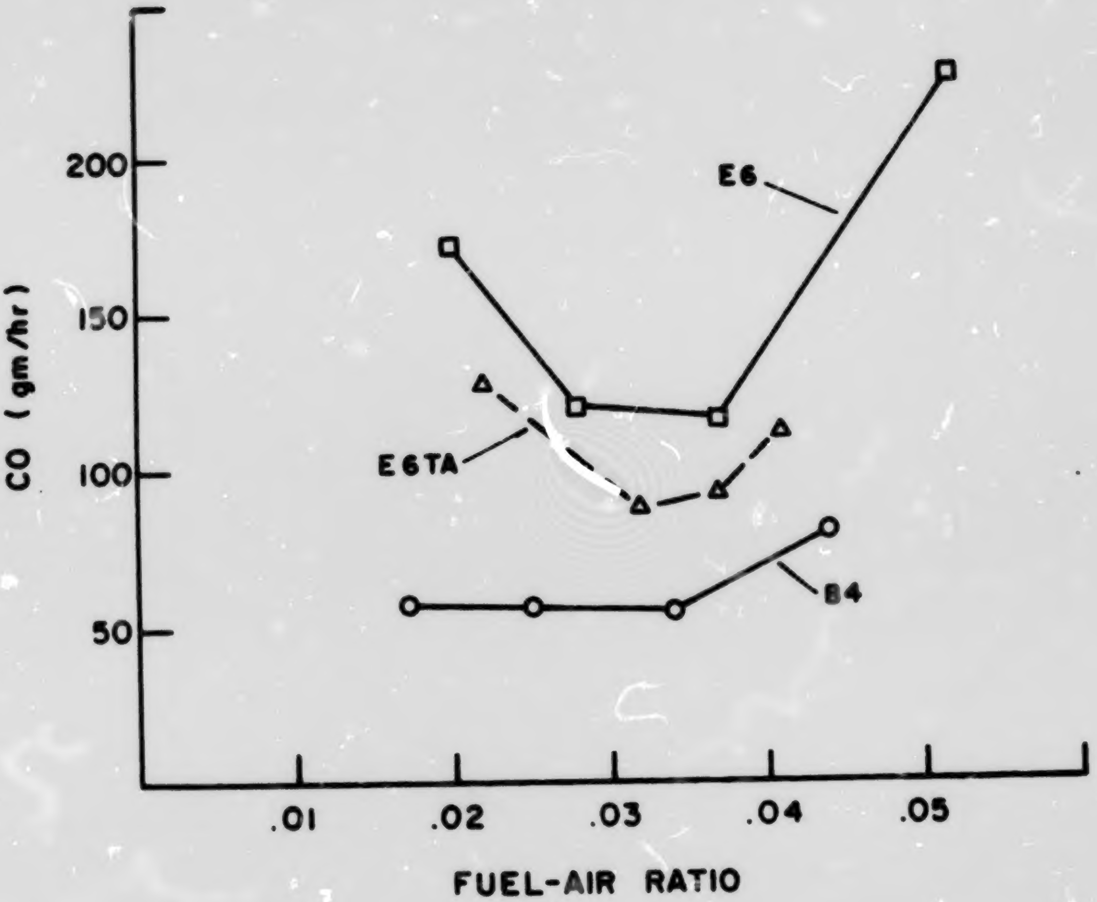


FIGURE 43

CO emissions (gm/hr) versus F/A ratio

(Source: Reference 453)

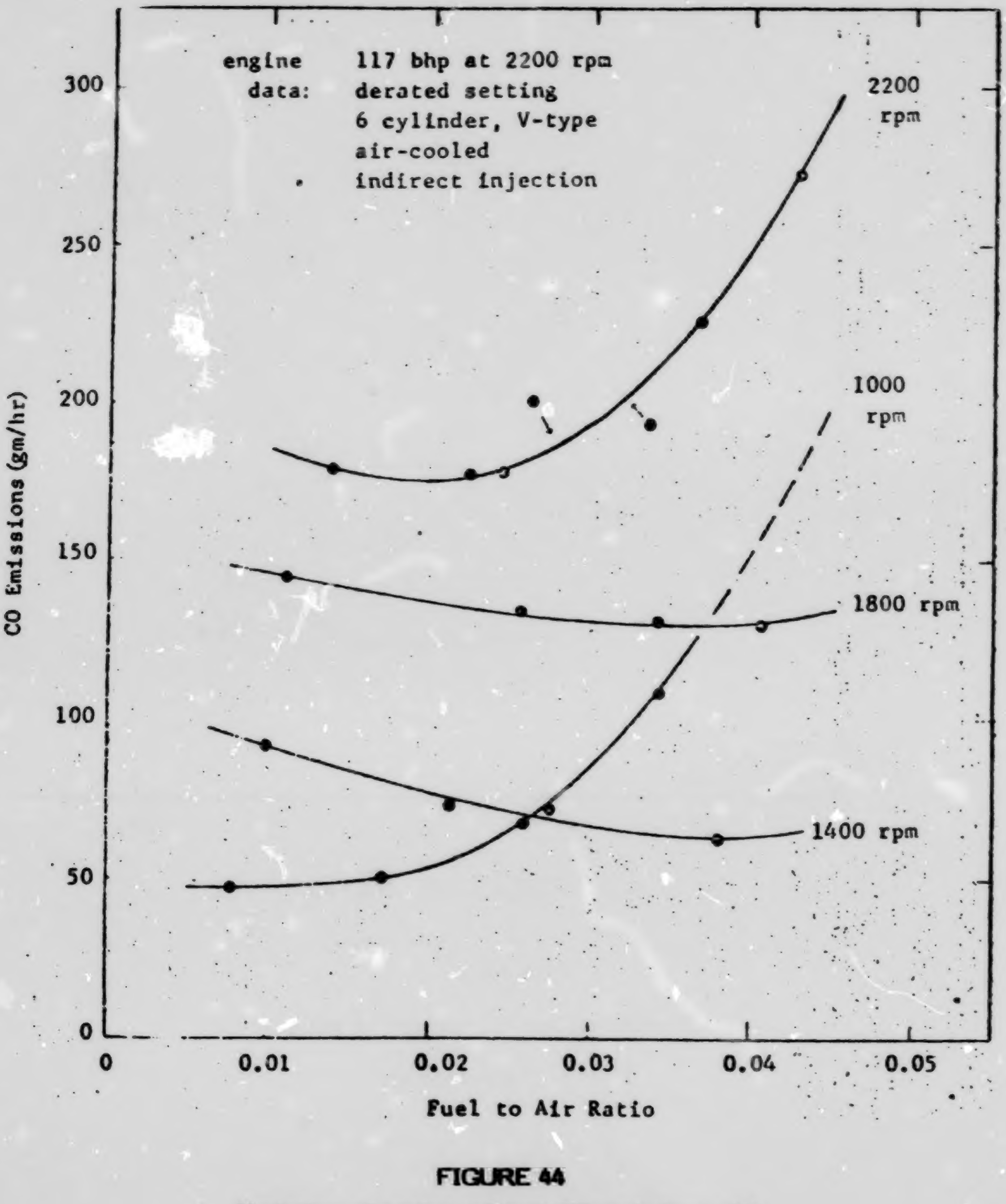


FIGURE 44

Variation of engine exhaust CO emissions with F:A ratio and speed

(Source: Reference 56)

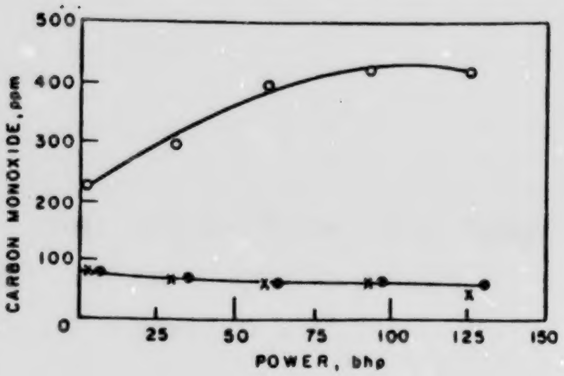


FIGURE 45

CO emissions from an IDI engine without catalytic converter (o) and with converter (x, ●)

(Source: Reference 65)

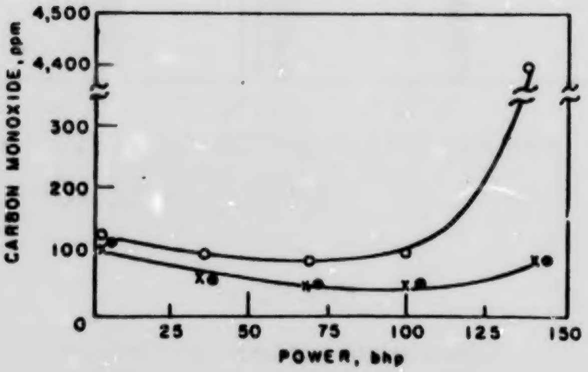


FIGURE 46

CO emissions from a DI engine without catalytic converter (o) and with converter (x, ●)

(Source: Reference 65)

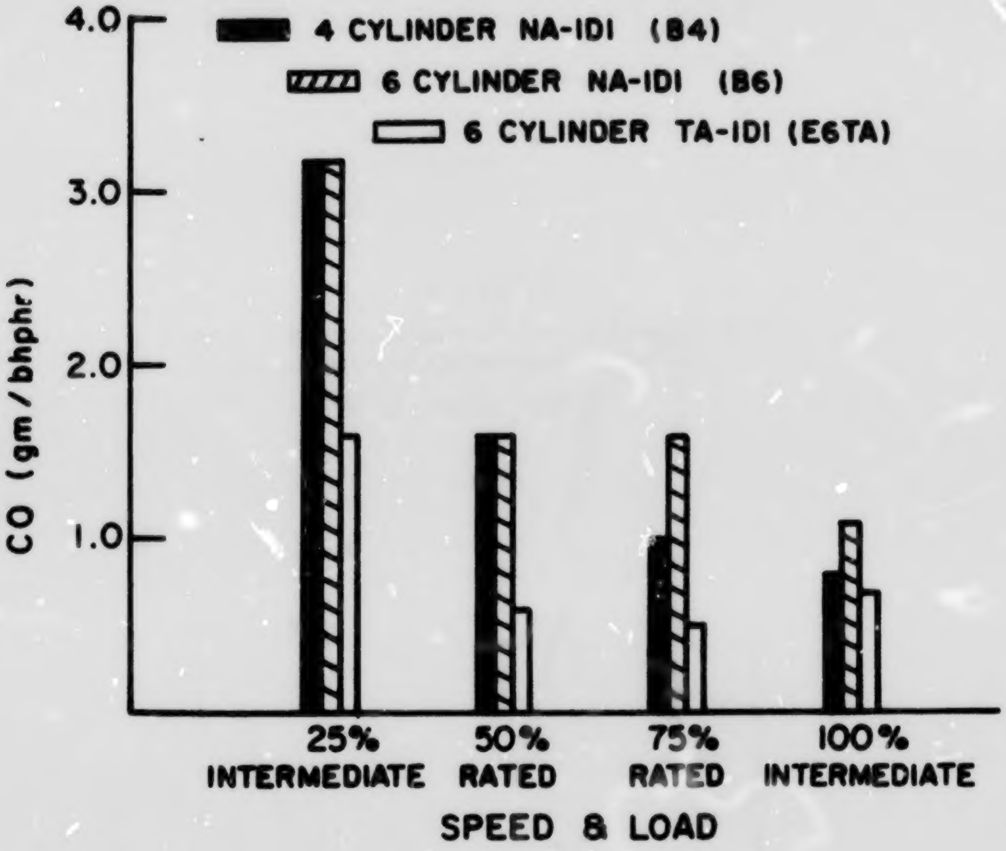


FIGURE 47

CO emissions versus speed and load
(4 segments of 13-mode cycle)

(Source: Reference 453)

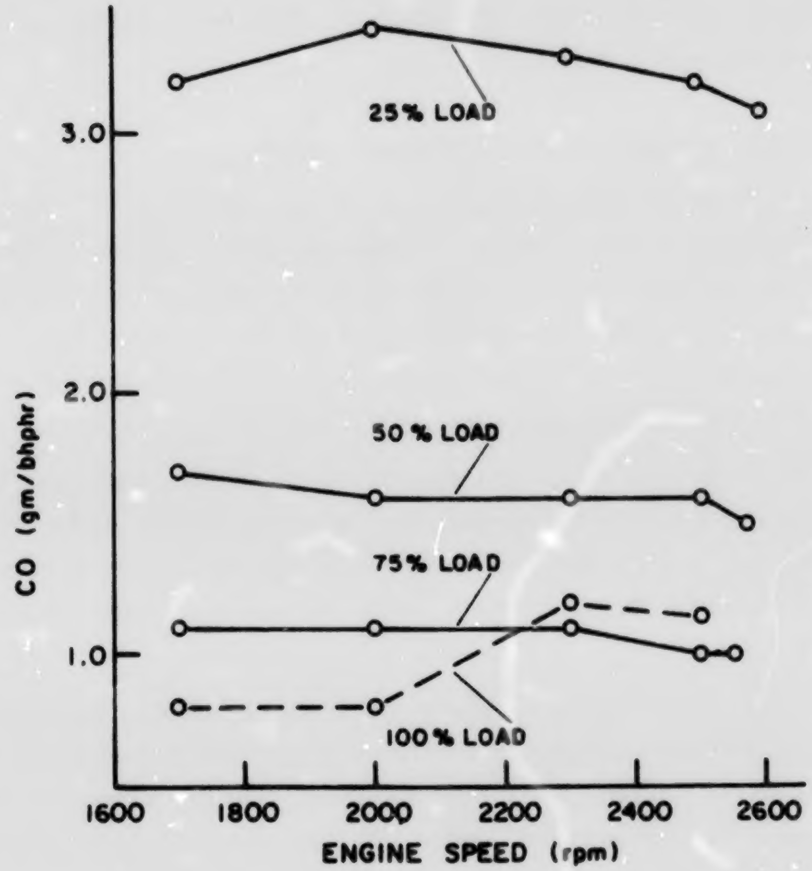


FIGURE 48

CO emissions versus speed and load (NA engine)

(Source: Reference 453)

- In summary, the above data show that carbon monoxide emissions are less with turbo after-charged engines, except when they are running at full load where the CO output is about the same for both naturally-aspirated and turbocharged after-cooled engines. The NIOSH results also demonstrate that the number of engine cylinders has almost no effect on the IDI CO brake-specific emissions (g/bhp-hr) and that CO output, as g/bhp-hr, decreases considerably as the horsepower increases.

3.4.4 Nitrogen Oxide and Nitrogen Dioxide

In general, the formation of NO_X occurs in the lean flame region of a diesel combustion chamber, and is related to combustion temperature. Although NO predominates, small amounts of NO_2 (less than 5% of NO_X), are also produced (56, 64, 66). NO_X formation is a function of rpm and load, and typical emission patterns for a Deutz-IDI, derated, V6 engine are given in Figure 49.

An inverse relationship between the F:A ratio and the NO concentration in the exhaust is evident (Figure 49). As the F:A ratio approaches the derated maximum of about 0.04 - 0.05, the NO emissions level off at higher speeds and drop significantly at low rpm's (Figure 49).

The comparative studies completed by NIOSH (453) found that NO emissions are higher from turbocharged aftercooled diesel engines than from naturally-aspirated diesels and that the number of cylinders has no effect on the NO output of IDI engines. Of significance is the finding that the brake-specific NO output (g/bhp-hr) averaged over segments 3, 6, 9 and 10 of the EPA 13-Mode Cycle were higher than NO levels at peak torque or at rated load/speed. Brake-specific NO emissions decreased with increasing fuel:air ratio up to an F/A of 0.050. NO mass output (g/bhp-hr) decreased as the engine horsepower increased.

Mogan and co-workers (56) found NO_2 levels in the exhaust to be generally insignificant. The few instances where NO_2 was quantitated were at idling or low power conditions, and the maximum

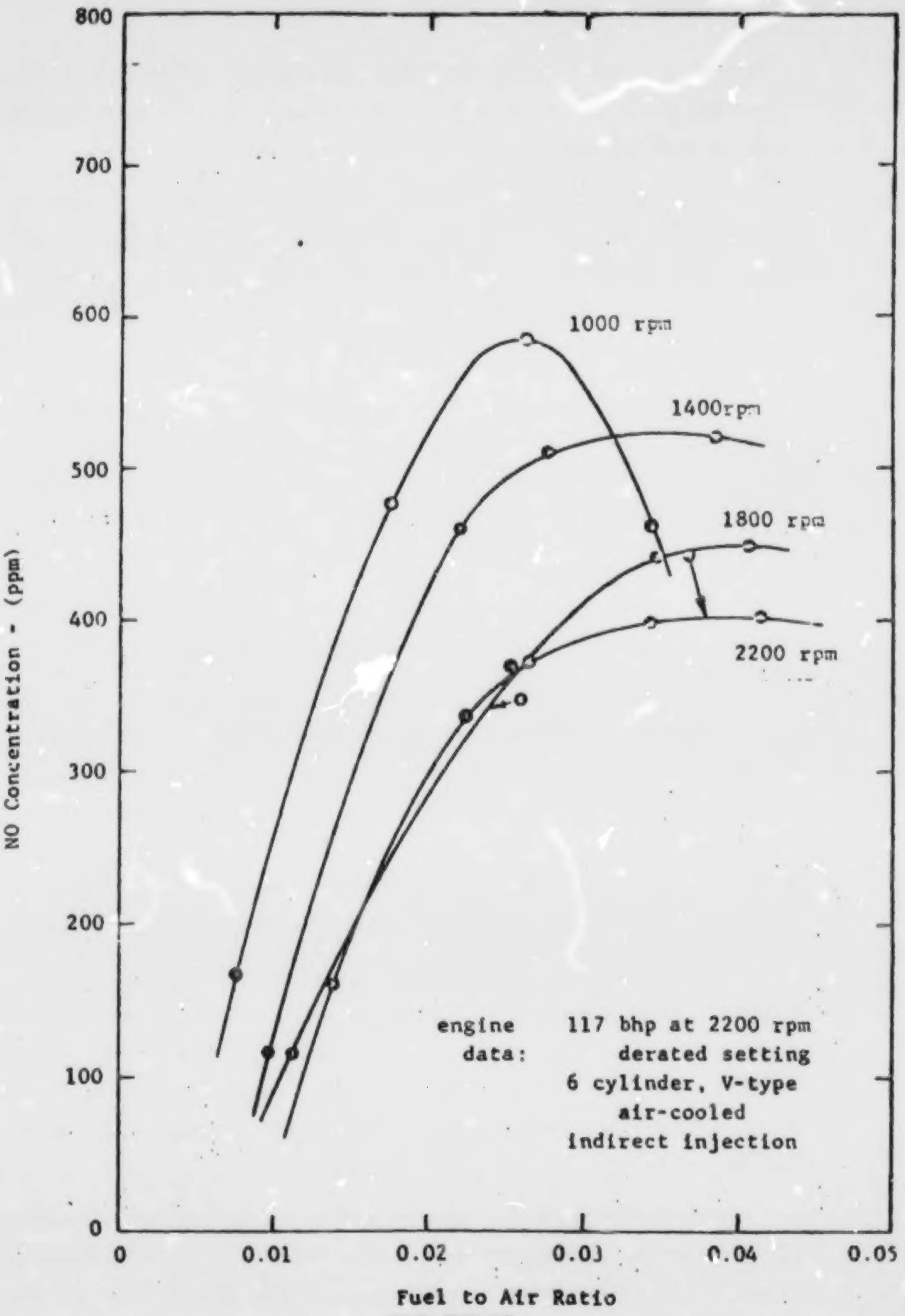


FIGURE 49

Variation of engine exhaust NO concentration with F:A ratio and speed

(Source: Reference 56)

detected NO_2 was 45 ppm. Out of 23 measurements taken at various operating modes, NO_2 was only found 5 times. The ORF also found in their studies (57) that all NO_X was present as NO.

Generally, at all loads IDI engines emit less NO_X than DI engines. For example, Stewart et al (67) found an IDI-derated, swirl-type engine to emit a total of 641 ppm of NO and zero NO_2 over a 700-foot haul distance, whereas a DI engine resulted in 1031 ppm of NO and 1.4 ppm NO_2 over the same distance.

Bone and Wheeler (61) reported up to 100 ppm NO_2 generated in exhaust by 6-cylinder, swirl-chamber and DI engines. Peak NO_2 occurred at low (15%) load and the concentration in exhaust decreased with increasing loads "as expected from thermodynamic considerations."

The conversion of NO to NO_2 in the mine atmosphere, which could contribute significantly to the total NO_2 exposure in a mine, is discussed in Section 3.9.2

Marshall (68) measured NO_2 emissions from the five different engines listed in Table 27. Figure 50 shows the fraction of the total NO_X which was in the exhaust for a 140 hp DI 4-stroke engine. According to the author, all other engines "exhibited similar dependence of NO_2/NO_X on load". For example, see Figure 51 which depicts similar data for a 150 hp, 4-stroke precombustion (IDI) engine.

NIOSH evaluation of various engines provided the data shown in Figures 52 and 53 in which the NO emissions versus speed and load are given for a naturally-aspirated engine and a turbocharged, after-cooled engine, respectively. Figure 54 shows the differences in nitric oxide emissions with speed and load for NA-IDI engines as well as an IDI-TA engine (453).

Figure 55 and 56 (453) provide NO emission data versus fuel:air ratios presented in mass emission g-bhp/hr and g/hr for comparative purposes for a 6-cyclinder NA-IDI engine (E-6), a 6-cylinder TA-IDI engine (E-6TA) and a 4-cylinder NA-IDI engine (B-4).

In 1975, Pitts and co-workers (14) reported the direct mutagenic activity of organic extracts from samples of ambient

TABLE 27

Engine Specifications

Engine	Max. power, bhp	Features
1	140	2-stroke cycle, direct-injection
2	50	4-Stroke cycle, direct-injection
3	100	4-Stroke cycle, precombustion chamber
4	125	4-Stroke cycle, precombustion chamber
5	150	4-Stroke cycle, precombustion chamber

(Source: Reference 68)

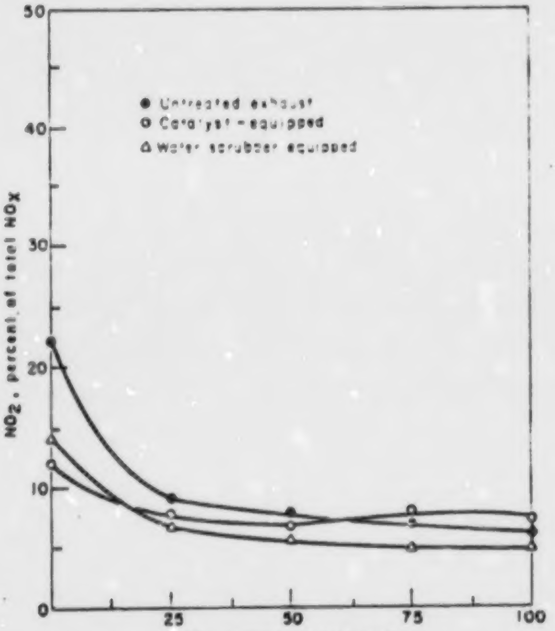


FIGURE 50

Effect of exhaust treatment on NO_2 fraction — DI engine, rated speed

(Source: Reference 68)

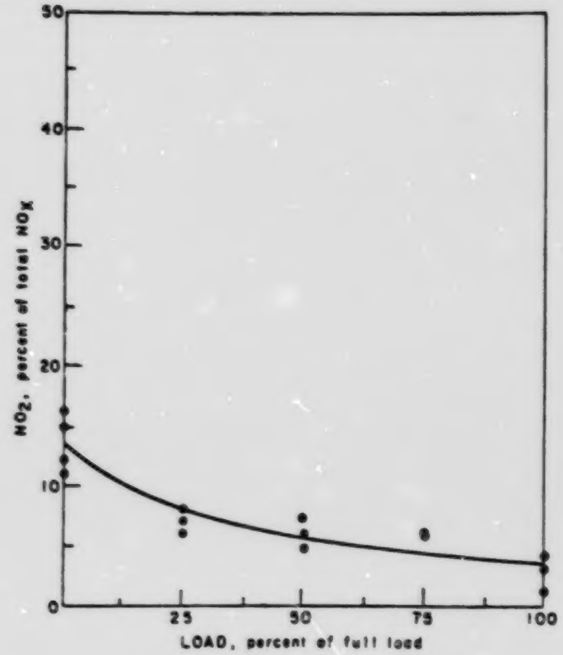


FIGURE 51

NO_2 fraction of total NO_X versus engine load -- IDI engine

(Source: Reference 68)

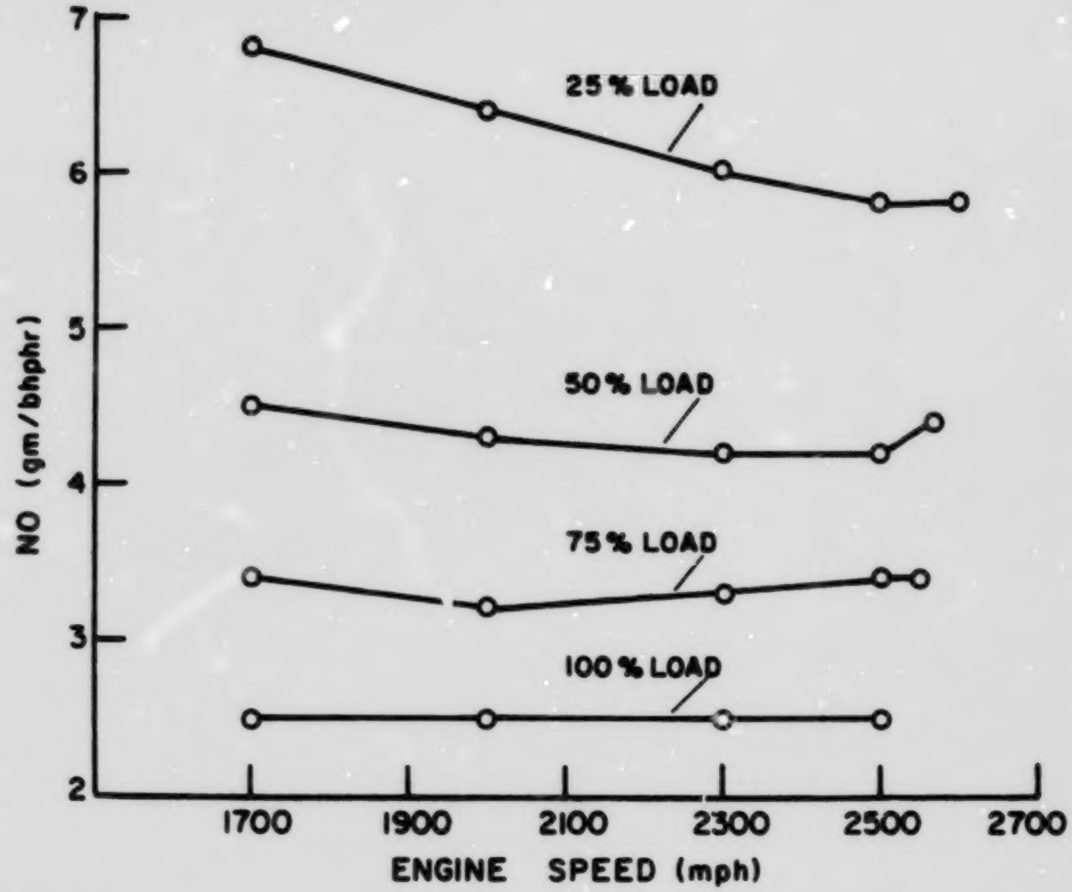


FIGURE 52

NO emissions versus speed and load (NA engine)

(Source: Reference 453)

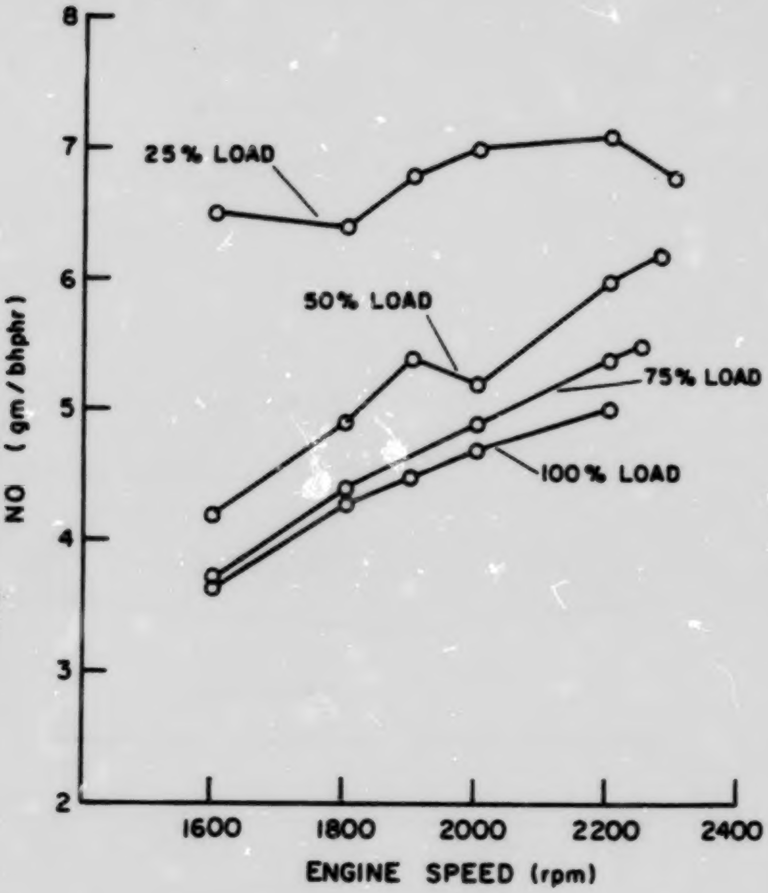


FIGURE 53

NO emissions versus speed and load (TA engine)

(Source: Reference 453)

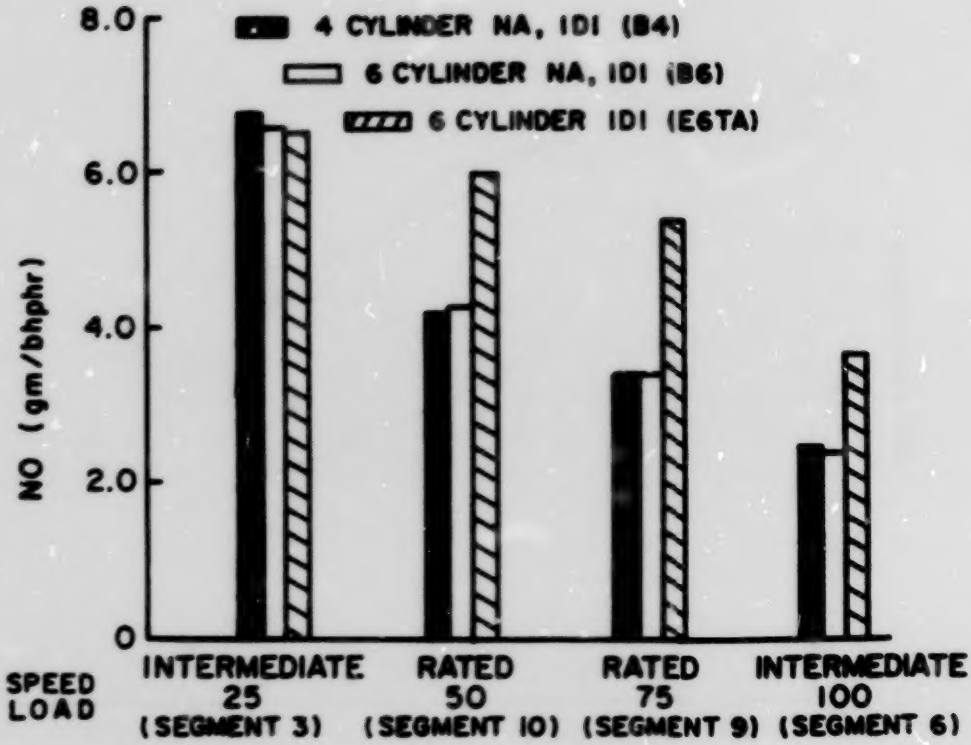


FIGURE 54

Nitric oxide emissions versus speed and load
(four segments of 13-mode cycle)

(Source: Reference 453)

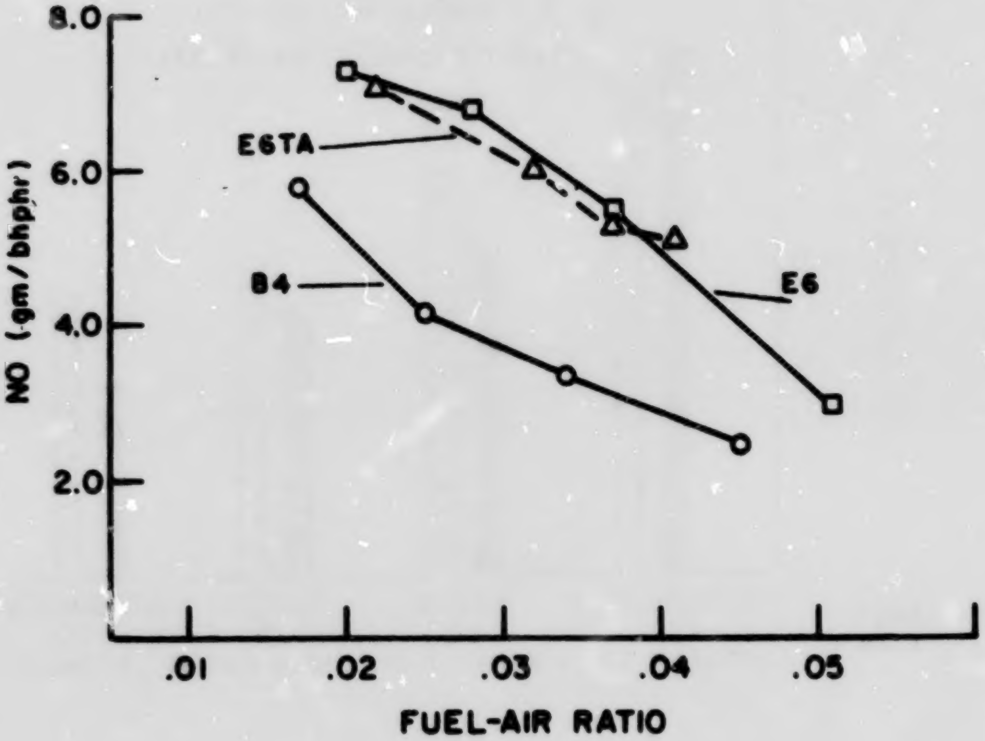


FIGURE 55
NO emissions (gm/bhphr) versus F/A ratio
(Source: Reference 453)

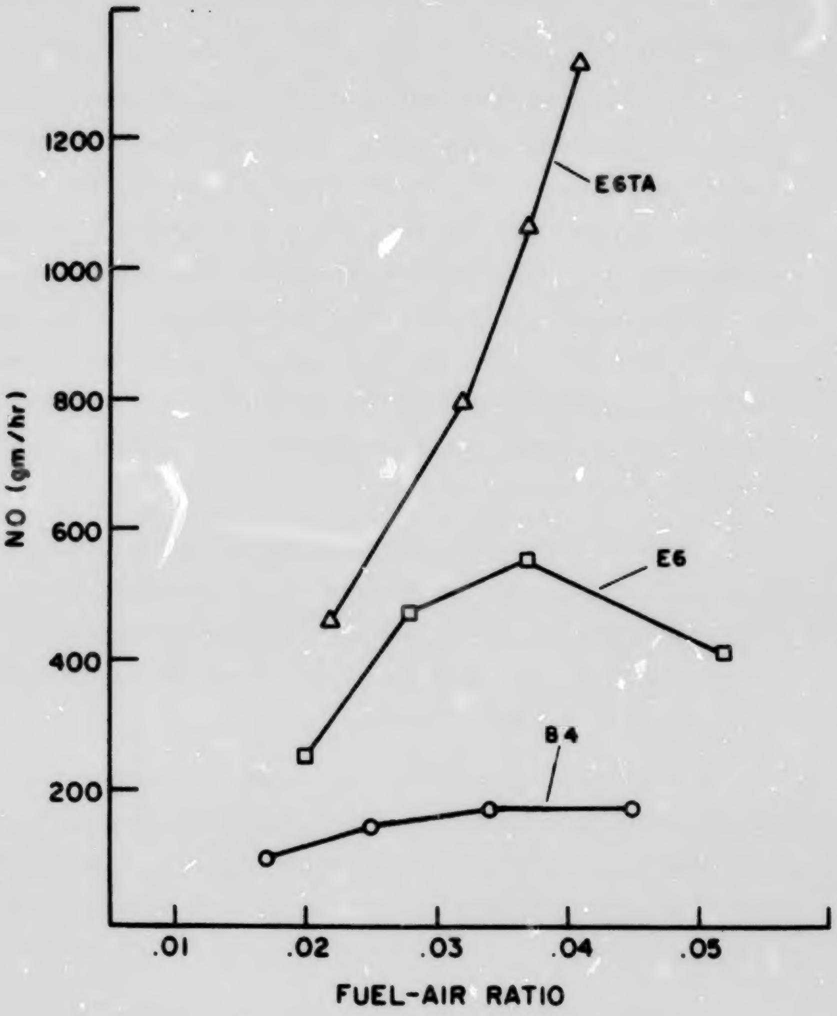


FIGURE 56
NO emissions (gm/hr) versus F/A ratio
(Source: Reference 453)

aerosols collected throughout Southern California which led to the investigation of the reactions of benzo(a)pyrene (BaP) deposited on glass filters with gaseous components of photochemical smog and with NO_2 and O_3.

Thin layer and high pressure liquid chromatographic techniques showed that a number of BaP derivatives were readily formed in both types of experiments. In contrast to BaP, which requires microsomal activation to produce mutagenic activity in the Ames Test, mononitro and oxygenated derivatives were readily formed upon exposure of the BaP coated filters to 0.25 ppm of NO_2 for 1 hour in the presence of about 10 ppb HNO_3. The resulting products were directly mutagenic and did not require activation. The exposure of 0.25 ppm NO_2 and 10 ppb HNO_3 for 1 hour corresponded to the California Air Quality Standards for these pollutants.

Although the topic of the mutagenicity of nitrated derivatives of polyaromatic nuclear hydrocarbons will be discussed in more detail under health effects, the following may assist in setting the "scene" for this important area relevant to the potential health risks which may be attributed to diesel exhaust.

It is interesting that a directly mutagenic mononitro derivative, 3-nitroperylene, was formed when ppm levels of NO_2 and 10 ppb HNO_3 in air was passed over perylene similarly deposited on a glass fibre filter. The parent perylene is an isomer of BaP, but is a much weaker mutagen than BaP.

A "direct mutagen" is defined as one not requiring microsomal activation. The author was aware of the possibility of increased sensitivity of the intracellular nitrogen reductase enzymes of the original test and the potential for false positives.

The nitration of BaP in simulated atmospheres appeared to be catalyzed by nitric acid. Spectroscopy studies have shown that there is ample HNO_3 in ambient photochemical smog to serve this function. Presence of HONO and NO_3 in ambient air has been confirmed and it is possible that these co-pollutants of particulate organic matter (POM) may also react with BaP and other reactive PAH's.

The authors emphasized that assessments of the health effects of exhaust emissions from diesel engines must take into account not only the chemical and physical characteristics of the emissions of particulate organic matter from the tailpipe, but also the chemical and physical transformations which may occur in the ambient air or during collection before and during receptor impact. Transformations of particulate organic matter by co-pollutants might occur in primary exhaust streams, transport in polluted atmospheres, on filters during the act of collection and possibly in situ in the lungs.

The authors noted that it is a common assumption that if the PNA levels in exhaust are reduced by certain control techniques, such as a catalytic converter, that the health impact of the POM will be lessened. However, this may not be so, especially if the catalyst is aged or inefficient. Thus, if BaP reacts or disappears from the system, new species of PAH may be formed which are more mutagenic and/or more carcinogenic than the orginal BaP. In this instance the health effect could be worse than before the treatment of the POM by the oxidizing catalyst.

Diesel particulate is in the respirable range and can remain in the atmosphere and be transported and transformed over a period of hours and possibly even days. Consequently, interactions with other chemical species in the environment must be considered. Thought must be given to the different consequences of injecting diesel emissions into clean air or into light, moderate or heavily polluted air, or even into heavy sulphur laden smog or photochemical smog, such as occurs in California and London, England. It was suggested that when evaluating the chemical and physical transformations of polyaromatic hydrocarbons, nitric and nitrous acids, formaldehyde, formic acid and the nitrate radical (NO_3) should be considered. Recent work by the authors found the latter reacts rapidly with phenol and cresol isomers, as well as with olefins, and it was postulated that it may also react with PAH.

The authors pointed out the need to precisely define the term "dose of diesel POM". Consideration must be given to the fact that their properties may be substantially modified by, for instance, the sampling process itself. The problem of filter artifacts has arisen before. For example, reactive polycyclic aromatic hydrocarbons such as BaP, are transformed on conventional high volume glass filter fibres by gaseous pollutants present in the ambient air. They may also react with SO_X, such as SO_2, SO_3, H_2SO_4, but these systems have not yet been examined.

It was also argued that if PAH's react on filters with atmospheric pollutants such as nitrogen dioxide, they might also react in situ after deposition in the lung with such gaseous co-pollutants dissolved in lung fluids. Thus, even laboratory animal exposure studies which were designed to clarify health effects of emissions from diesel POM may be incomplete unless the studies simulates the potential synergism between the POM and its co-pollutants.

Although this work was interesting and somewhat provocative, much of it was speculative and not based on experimental data. The influence of smog, ozone, etc., are, of course, not relevant to the underground environment.

Wheeler, Hear and McCauley (69) presented pollutant levels measured in a U.S. coal mine using diesel teletrams as rock transport vehicles. The contaminant exposure of various workers at various locations in the mine were presented. Of most importance were the exposures for the continuous miner operator and the teletram operator breathing zones. Table 28 provides data for respirable dust, nitrogen dioxide, cyclohexane extractable fraction and a few other components of lesser interest. Particulate levels were probably more indicative of the diesel emissions. The total aldehyde concentration was also considered to be indicative of diesel contamination and was found to be highest for the breathing zones of the continuous miner-operator and the teletram operator. NO_2 data were based on an 8-hour time-weighted average (TWA) so that brief excursions would not be recorded. However, the highest TWA exposure was to the continuous miner and teletram operators who received geometric means of 0.24 and 0.21 ppm, respectively. These are well within current ACGIH limits.

TABLE 28

Location	No. of Samples	Geometric Mean	Geometric Standard Deviation	Range	Coefficient of Variation
RESPIRABLE DUST (mg/m^3)					
Roof Bolt Operator	3	0.91	1.97		
Teletram Operator	7	0.58	1.68		
NITROGEN DIOXIDE (ppm)					
Roof Bolt Operator	10	0.03	2.80	0.01 - 0.17	
Teletram Operator	15	0.21	1.52	0.08 - 0.45	
CYCLOHEXANE EXTRACTABLE FRACTION (mg/m^3)					
Roof Bolt Operator	2	0.07	1.10		96%
Teletram Operator	7	0.04	2.93		72%
TOTAL ALDEHYDES (ppm)					
Roof Bolt Operator	2	0	1.00	0	
Teletram Operator	3	11	1.56	7 - 17	

(Source: Reference 69)

Lenner and co-workers (70) studied the influence of a Volvo diesel particulate on the oxidation of nitric oxide to nitrogen dioxide at different conditions of load, temperature and dilution. The formation of NO_2 from NO could be described by the formula;

$$-(d(NO)/dt) = k(NO)^2(O_2).$$

The rate constant had a negative temperature dependence, especially at higher dilution rates. The reaction was enhanced by the presence of diesel particles, although the latter effect was not as strong as the catalytic effect of street and wall surfaces reported by other investigators. Spectroscopic data indicated that unexposed samples gave the same kind of spectra as samples which had been exposed to NO_2 and UV light, indicating that the reaction was not light-dependent.

The Volvo Corporation (71) reported the results of a study in which the rate constant for the oxidation of nitric oxide to nitrogen dioxide was calculated under various conditions of temperature and dilution, for a Volvo passenger diesel engine. It was found that the rate of conversion was 1.55 ($m^{-2} \times sec.^{-1} \times 10^{-4}$) in the absence and 1.80 in the presence of diesel particulate. It was found that the effect of diesel particulate on the conversion of NO to NO_2 was greater than that of inorganic aerosols, such as ammonium sulphate, but appeared to be minor compared to the catalytic effect of street and house wall surfaces. The authors pointed out that diesel-powered cars emit upwards of 250 ppm of NO_X of which as much as 30% may initially be in the form of NO_2. Formation of additional NO_2 is enhanced by low temperatures and by the availability of suitable surfaces which catalyze the conversion of NO to NO_2. Particulate emissions from diesel engines may play at least a minor part in this respect. Their studies showed that nitrogen-containing species attached to diesel particulate, particularly those exposed to NO_2 and UV light.

3.4.5 Sulphur Oxides

The quantity of SO_2 emitted in diesel exhaust is directly related to the sulphur content of the fuel, as was discussed in Section 3.3.4. With a TLV of 0.25 ppm, SO_3 levels represent a pollutant of serious concern.

Marshall (65) studied the formation of SO_3 and H_2SO_4 and found that the emission of SO_3 from an IDI, 4-stroke engine increased dramatically with heavy load conditions (Figure 57) when the exhaust was run through a catalytic converter. This effect appeared to be related to exhaust temperature (Figure 57). The level of SO_3 was directly related to the fuel sulphur concentration (Figure 58) and accounted for about 1/3 of the total fuel sulphur.

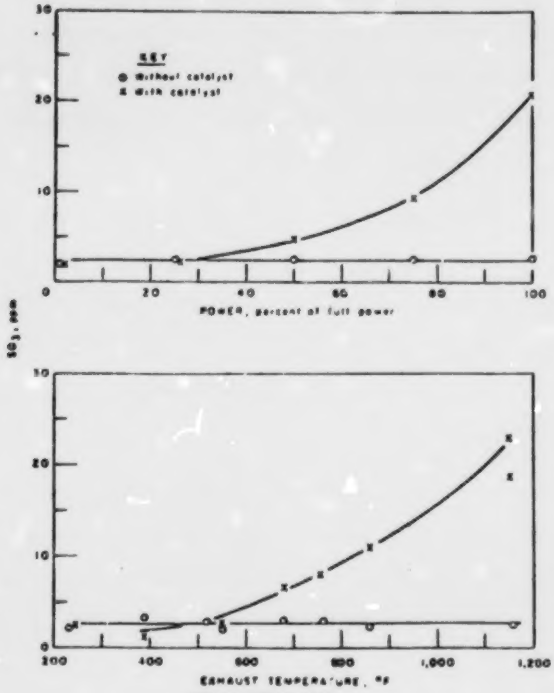


FIGURE 57

Effect of catalyst on SO_3 emissions -- IDI engine
With catalyst (x); Without catalyst (o)

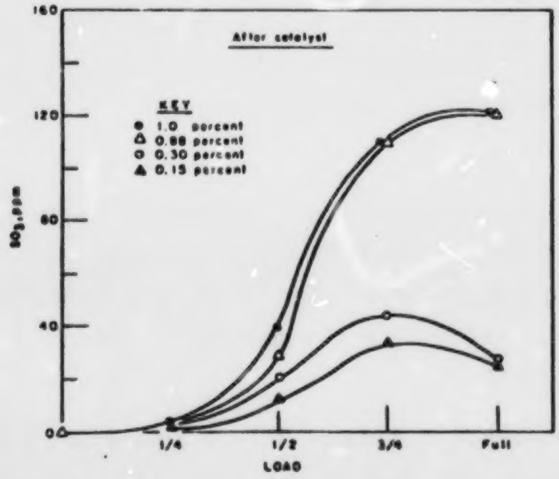


FIGURE 58

Effect of fuel sulphur level on SO_3 emissions - IDI engine

(Source: Reference 65)

Clearly, a reduction in sulphur oxide emissions can be achieved by reducing the sulphur content of diesel fuel.

In Ontario, diesel fuel used in underground diesels cannot contain more than 0.25% sulphur. Lowering this value to 0.1 - 0.15% could result in significant reductions in SO_2 emissions by engines equipped with platinum purifiers.

As is discussed in depth in Section 3.9.8, the measurement of ambient SO_2 levels may underestimate the true health risk of suphates due to absorption of SO_2 onto diesel soot, solution in water and the conversion of SO_2 to H_2SO_4. There is a need to more clearly define these interactions and their significance in terms of possible adverse health effects.

Ambient SO_2 levels in mine air may only represent about 25 - 50% of the total SO_2 present due to adsorption onto particulate and/or dissolution in water. It can be calculated that with a 0.25% S-fuel and a 9% CO_2 level, that the SO_2 should be 100 ppm. However, actual mine levels seldom reach these concentrations for the reasons indicated above (48, 72, 73).

3.4.6 Hydrocarbons

Both unburned and partially oxidized hydrocarbons can be found in diesel exhaust. In addition, decomposed and recombined fuel hydrocarbons are also present. The mixture of hydrocarbons is complex and includes aldehydes, ketones, phenols, as well as alkanes and alkenes, etc.

Although not particularly relevant to diesels used underground, data from Linnell and Scott's (12, 74) examination of exhaust from a motor coach-type engine using No. 2 diesel fuel, provides an indication of the complexity of the hydrocarbon emissions coming from diesel engines (Table 29).

According to Johnson (38) and others (75), the hydrocarbons are fuel-derived, with their qualitative and quantitative aspects being related to fuel injection and combustion chamber design. To a certain extent, hydrocarbon emissions can be controlled along with CO by use of catalytic converters and fuel injection changes (53).

TABLE 29

Hydrocarbon Content of Diesel Exhaust (ppm)

Compound	800 rpm	1,050 rpm	1,300 rpm	1,520 rpm	1,780 rpm
Acetylene	2.46	1.70	2.17	3.09	5.32
Ethylene	12.2	8.93	13.7	18.5	27.3
Propane	0.6	0.08	0.11	0.20	0.19
Propylene	3.18	3.96	4.67	5.48	8.55
n-Butane*	<0.05	<0.05	<0.05	<0.05	0.12
1-Butene	0.80	0.95	1.30	1.50	2.07
isoButylene	0.45	0.49	0.53	0.73	1.20
isoPentene	<0.05	<0.05	<0.05	0.19	0.08
2-Butene	<0.05	0.22	0.22	0.29	0.73
n-Pentane +	0.10	0.14	0.13	0.08	0.47
1,3-Butadiene +#	0.64	0.57	1.01	1.12	1.51
1-Pentene +	0.52	0.69	0.91	1.08	1.43
Total Olefin +++	1.46	2.00	2.12	3.11	4.33

* The symbol <0.05 indicates the compound is present but too low for measurement.

+ Calculated as propane.

# There may be some 2,2-dimethylbutane present which overlaps the 1,3-butadiene.

+++ Colorimetric method. This method is nonspecific for C-4 and higher olefins and is insensitive to acetylene, ethylene and propylene.

(Source: Reference 10)

Total hydrocarbon emissions (as carbon) from IDI and DI engines, with and without catalysts, are shown in Figure 59 (65). Both the monolithic (line A) and pelletized (line B) converters were effective in reducing total carbon output. However, Marshall (65) found that odour levels were significantly reduced only by the pelletized purifier.

This latter point concerning odour was raised as it is thought that the partial oxidation products, such as aldehydes like acrolein and formaldehyde (67, 74, 76, 77) are the main contributors to diesel exhaust odour.

The effect of engine load at rated speed on the exhaust content of total hydrocarbons is given in Figure 60 (78). This data was derived using a DI, 4-cycle, turbocharged engine.

Smoke (black) or soot is also related to the amount of unburned carbon emitted by diesel engines. It is composed of particulate carbon of 0.4 - 1.0 u diameter. Smoke is formed when local temperatures are sufficiently high to decompose the fuel, but insufficient oxygen is present to permit complete combustion (38).

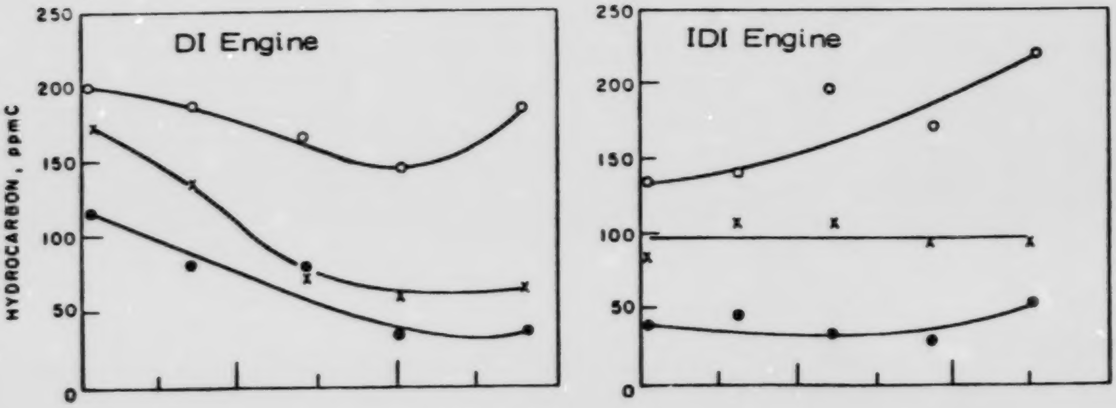


FIGURE 59

Hydrocarbon emissions from an IDI and DI engine without catalytic converter (o) and with converter (x, •).

(Source: Reference 65)

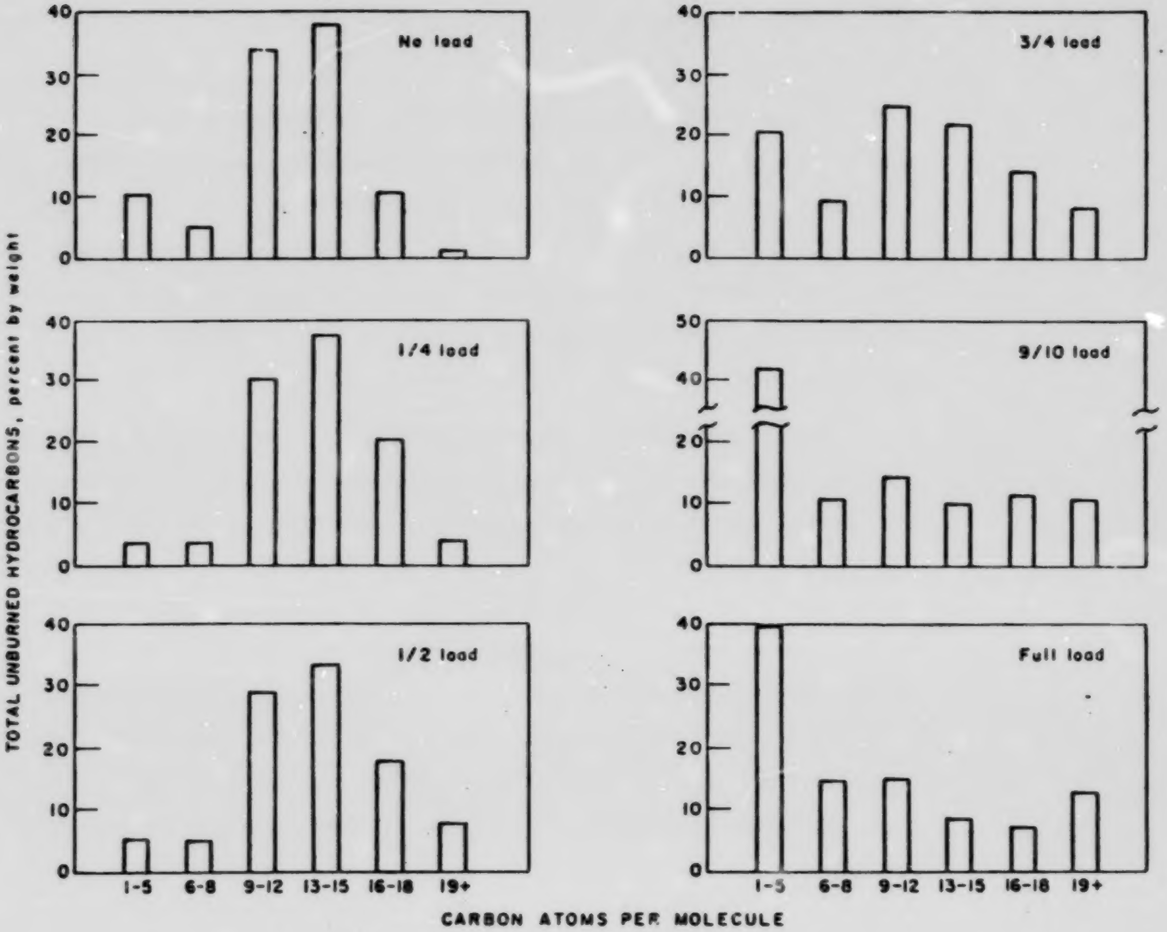


FIGURE 60

Effect of engine load on exhaust hydrocarbon distribution (DI, 4-cycle, turbocharged engine)

(Source: Reference 78)

3.4.7 Particulates

As with other sections in this document, the inter-disciplinary research by many groups into diesel particulate, PAH's and toxicity makes it difficult to confine discussions to a particular section heading, such as particulates and PAH's, etc. Therefore, overlap with these sections has been unavoidable and it is recomended these topics not be reviewed in isolation but together, thus preserving continuity and leading to a better understanding of a large and comprehensive body of work.

From a health risk point of view, particulate emissions from diesel engines are of particular concern. Because diesel particulate is composed primarily of "soot" or carbon particles, it is thought that diesel particulate provides a tenacious site for adsorption of gaseous and non-gaseous environmental constituents, including radon daughters, and possibly even radon itself, as well as gases such as SO_2 and NO_2, polycyclic hydrocarbons and acid aerosols.

It is necessary to quantitate the amount of particulate emitted during diesel use, as well as to delineate its spatial properties, particularly as related to respirability. Obviously, diesel particulate has the potential of acting as a carrier of noxious and toxic substances to the pulmonary system. Germane to this latter point is the determination of how tenaciously any particular substance is bound by diesel particulate. If binding is "loose", the adsorbed substance may be readily "eluted" off the particulate matter in the lungs, thus permitting expression of its toxic properties. On the other hand, if the toxic component is "tightly" bound to the diesel carbon, then it may remain on the particulate in the lungs and thus be "non-toxic". Although definitive answers to these questions are not available, various sections of this report will address the problem and provide some insight and points for debate.

Several detailed treatises are available on sampling and measurement technology for various types of particulates (for example, references 21, 79 and 80). Additional details of measurement methods for diesel particulate were published by Carlson & Johnson (37).

Several instruments have been developed for measuring exhaust and atmopsheric levels of dust and particulate, and will be discussed in more detail in Section 3.9.6. For orientation purposes, the method most generally employed to measure diesel particulate involves permitting the exhaust to flow through a series of filters, usually made of glass fibres. The filters are loaded in an instrument, such as an Anderson impactor, which has a series of plates with differing porosity which are designed to size particles from 20 um to 0.4 um. A glass fibre filter will trap particles less than 0.4 um.

An alternative method is to remove larger, non-respirable particles using an elutriator unit. A schematic drawing (Figure 61) of the sampling system used by Stewart and co-workers (62) depicts the manner in which respirable and total particulate can be evaluated. All particles were trapped on glass fibre filters of appropriate porosity.

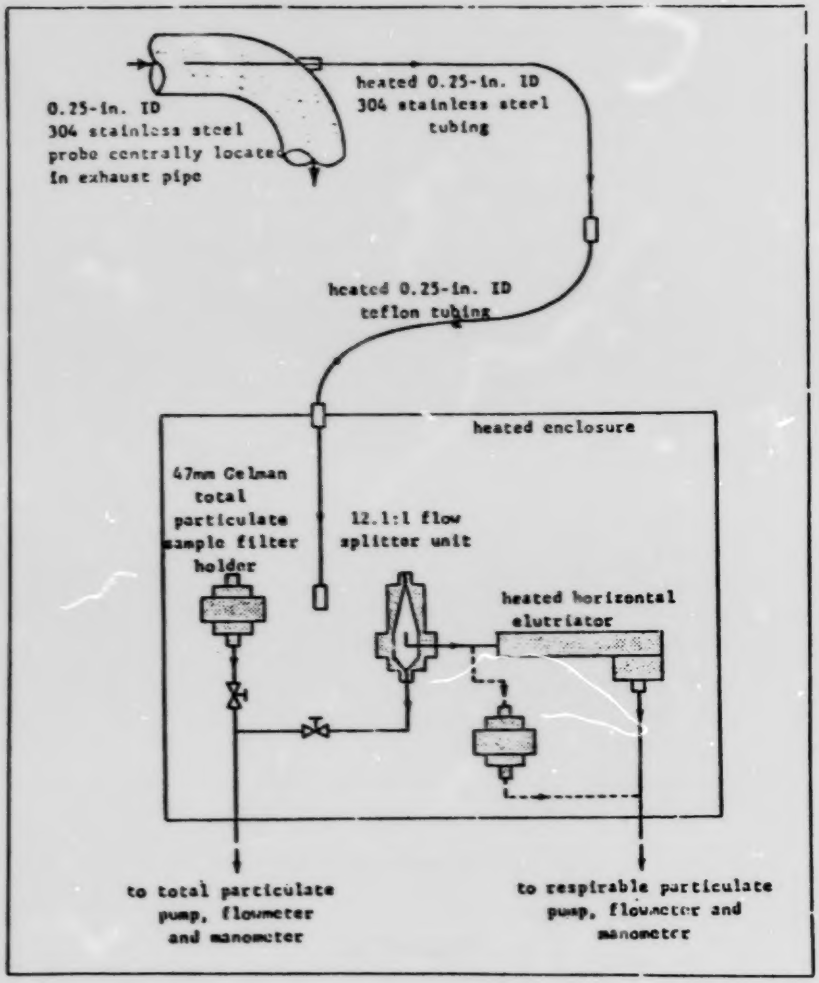

0.25-in. ID
304 stainless steel
probe centrally located
in exhaust pipe
heated 0.25-in. ID
304 stainless steel
tubing
heated 0.25-in. ID
teflon tubing
heated enclosure
47mm Gelman
total
particulate
sample filter
holder
12.1:1 flow
splitter unit
heated horizontal
elutriator
to total particulate
pump, flowmeter
and manometer
to respirable particulate
pump, flowmeter and
manometer

FIGURE 61

Schematic diagram of exhaust particulate sampling apparatus

(Source: Reference 62)

Using this latter equipment, Stewart et al (62) examined particulate emissions from both DI and IDI engines. At low F:A ratios, the IDI diesel emitted higher particulate levels than the DI engine. At rated power, and as the F:A ratio increased, both engines put out comparable amounts of particulate (see Figure 62). The authors suggested that for the usual underground diesels, at full engine load, a total particulate emission of 100 -150 mg/m^3 could be expected.

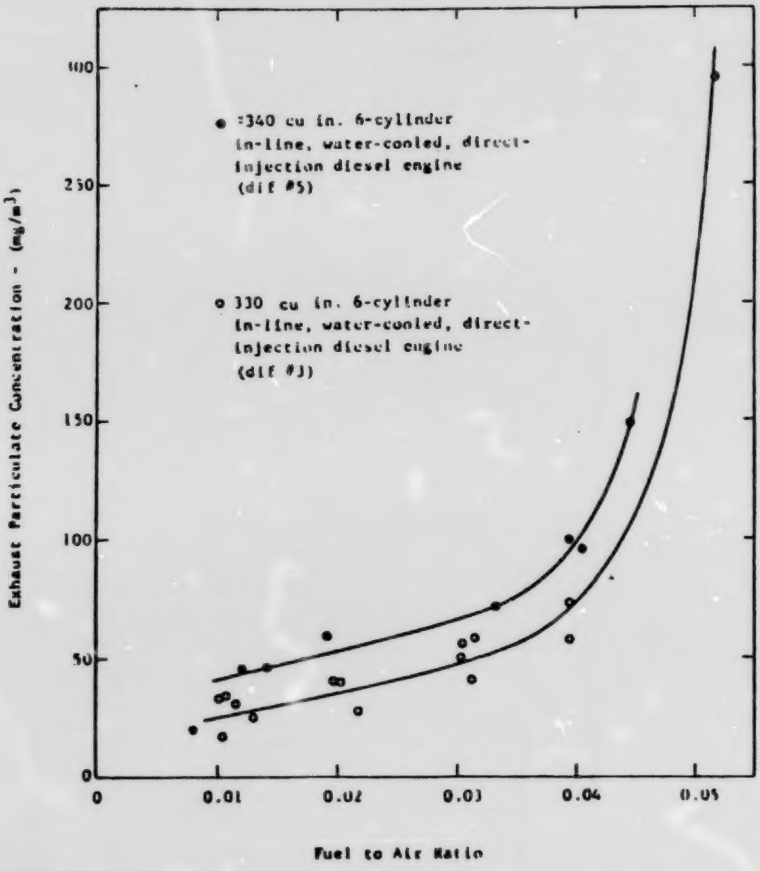


FIGURE 62

The variation of particulate concentration with F:A for two similar diesel engines using the same fuel

(Source: Reference 62)

A high percentage of diesel particulate was below 0.5 microns (u) in diameter and the investigators concluded that diesel particulate was essentially 100% respirable. A similar conclusion was reached by Breslin and co-workers (81) in studying particulate emissions from Caterpillar and Cummins engines. Further, they found that only sufficient ventilation would control particulate levels below 2 mg/m^3. In particular, concern was expressed that diesel particulate could add to the respirable dust in coal mines and make it more difficult to comply with the U.S. Bureau of Mines 2.0 mg/m^3 respirable dust standard. In general, the particulate levels found by Breslin et al (81) were higher (up to 250 mg/m^3) than those usually measured from Deutz diesels as used underground in Canada. Number 1 diesel fuel resulted in a significantly lower particulate mass output.

In reviewing the toxicity of each exhaust constituent, as related to the F:A ratio, it was determined that the particulate fraction was the largest single contributor to the toxicity of raw exhaust (62).

A typical engine used in a service vehicle, and normally operating with a F:A ratio of less than 0.03, would emit maximum particulate of about 60 mg/m^3.

In 1976, Vuk, Jones and Johnson (82) published a detailed account of the physical character of diesel particulate. Although the data are of considerable importance and relevance, it must be noted that particulate was generated using a DI engine not commonly found in underground use and having a relatively high (350 - 1100 ppm) hydrocarbon emission. A more general and detailed description of particulate formation and its physical and chemical character was recently released by Lipkea, Johnson and Vuk (21), and by Khatri, Johnson and Leddy (80). The complexity of the engine variables which can affect diesel soot formation given by Broome and Khan was diagrammed in reference 21 and is reproduced in Figure 63.

Diesel particulate was found to be composed of very small spherical particles which agglomerated to form larger masses (82). Individual particles ranged from 100 - 800 Å, and the agglomerates from 5 - 10 um (micrometers) in size. About 95% of the particles were less than 1 u in size and therefore largely respirable, thereby

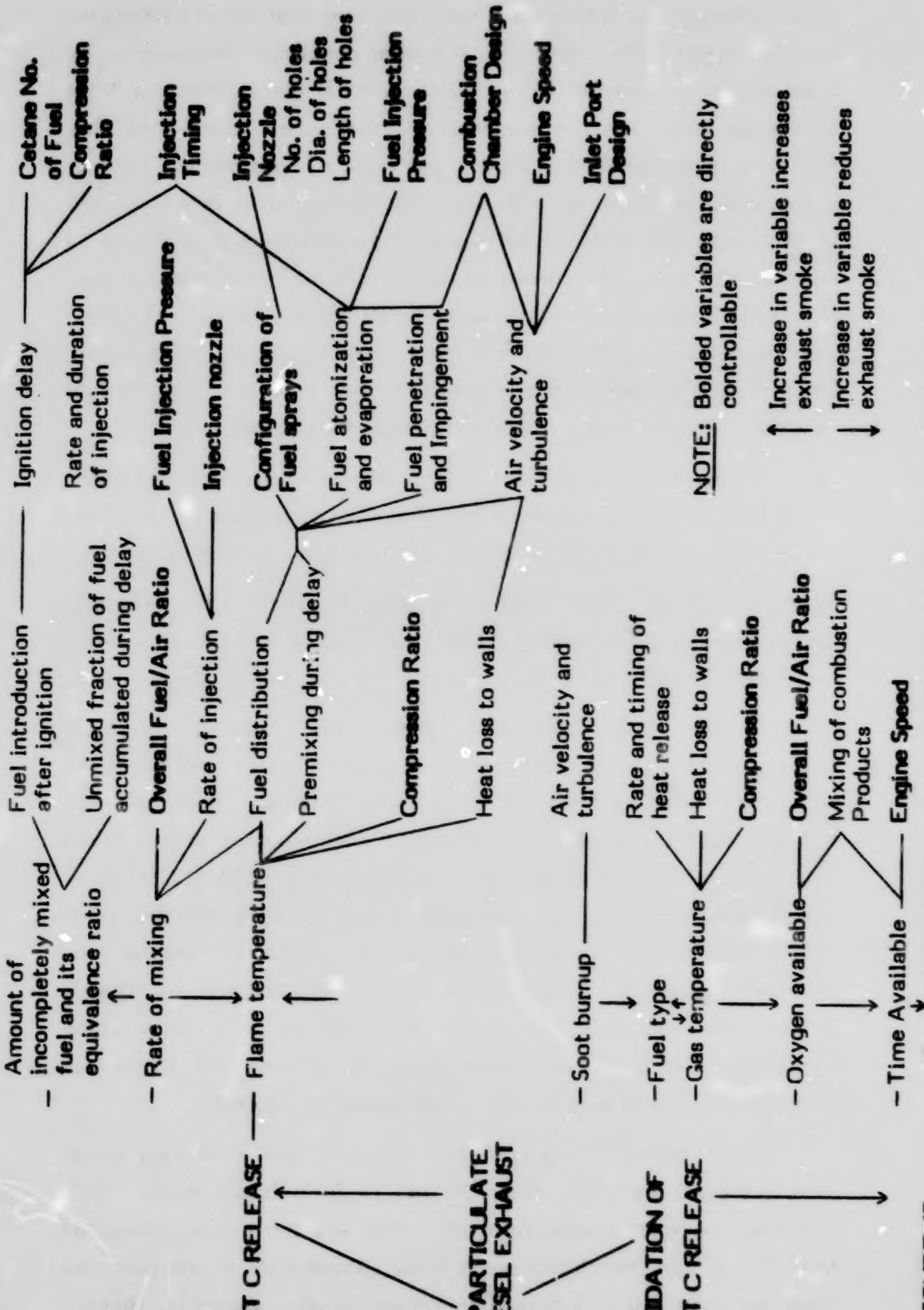

C PARTICULATE DIESEL EXHAUST
NET C RELEASE
OXIDATION OF NET C RELEASE
Amount of incompletely mixed fuel and its equivalence ratio
Rate of mixing
Flame temperature
Soot burnup
Fuel type
Gas temperature
Oxygen available
Time Available
Fuel introduction after ignition
Unmixed fraction of fuel accumulated during delay
Overall Fuel/Air Ratio
Rate of injection
Fuel distribution
Premixing during delay
Compression Ratio
Heat loss to walls
Air velocity and turbulence
Rate and timing of heat release
Heat loss to walls
Compression Ratio
Overall Fuel/Air Ratio
Mixing of combustion Products
Engine Speed
Ignition delay
Rate and duration of injection
Fuel Injection Pressure
Injection nozzle
Configuration of Fuel sprays
Fuel atomization and evaporation
Fuel penetration and Impingement
Air velocity and turbulence
Cetane No. of Fuel
Compression Ratio
Injection Timing
Injection Nozzle
No. of holes
Dia. of holes
Length of holes
Fuel Injection Pressure
Combustion Chamber Design
Engine Speed
Inlet Port Design
NOTE: Bolded variables are directly controllable
↑ Increase in variable increases exhaust smoke
↓ Increase in variable reduces exhaust smoke

FIGURE 63 **Summary of diesel engine variables affecting soot particular formation.** (Source: Reference 21)

confirming the reports discussed above (62, 81). Detailed discussions and electron micrographs, beyond the scope of this report, were given by Vuk et al (82) describing the size and structure of diesel particulate.

Carpenter and Johnson (83) examined 24 undiluted and diluted exhaust particulate samples using transmission electron microscopy which had an observed range of diameters of 70 -1200 angstroms (Å). The mean particle diameters in the undiluted and diluted exhaust samples were 479 and 436 Å, respectively. At lower temperatures of the diluted exhaust, evidence of hydrocarbon condensation was found. In some instances the condensed coating was so heavy as to completely obscure all detail of the aggregate versus agglomerate on which it is adsorbed and the differences between undiluted and diluted exhaust samples were quickly discerned.

For those modes exhibiting large concentrations of unburned hydrocarbon in the exhaust, there were correspondingly large amounts of condensed hydrocarbons on the particulate in the diluted exhaust. In some instances, notably Modes 2 and 3, coatings of condensed hydrocarbons were so heavy as to totally obscure the aggregate. The photomicrographs of particles in the undiluted and diluted exhaust provided experimental evidence that both separate liquid hydrocarbons and liquid hydrocarbon adsorbed on the solid particles exist in diluted diesel exhaust, but not in the undiluted exhaust. No significant size differences in the basic solid particles were found between undiluted and diluted exhaust samples.

It is worthwhile to re-emphasize that respirable particulate matter (RPM) measurement techniques do not differentiate between particulate matter originating from diesel exhaust and that from ore dust or oil mists. As pointed out by Carlson and Johnson (9), the analysis of RPM for carbon, hydrogen and nitrogen is useful in differentiating between diesel and ore RPM, but it is subject to considerable error, especially if the air contains significant quantities of carbon.

The problem of oil mists, which occur mainly at the working face, is ignored by these authors. Inco Metals Company consider the problem of identifying oil mists in RCD (respirable combustible dust) as being important. They consider that oil mist may constitute an "extremely high portion of an underground sample from both dieselized and non-dieselized areas."

Although this problem was not addressed by Carlson & Johnson (45), this may have been because oil mists may not have been a problem in the mines they examined, or because generally oil mists are generated during drilling operations prior to mucking out with LHD equipment and therefore the presence of significant oil mists during heavy diesel use is unlikely.

Using a duty-cycle designed to simulate LHD use in a "mucking" opeation, Stewart and colleagues (48) measured particulate emissions by IDI and DI diesels. A summary of their findings is given in Table 30.

When these values were integrated, the values for representative 200 and 700-foot runs were calculated and can be found in Table 31.

TABLE 30

IDI and DI Engine Particulate Emissions

		Particulate Emissions (mg/m^3)	
		IDI	DI
Phase	1 (preparing load bucket)	126.7	200
	2 (loading bucket)	163.9	130
	3 (hauling bucket)	80.0	65
	4 (preparing dump bucket)	113.0	200
	5 (dumping)	33.0	40
	6 (returning empty)	90.0	100

(Source: Reference 48)

TABLE 31

Integrated Particulate Emissions for DI and IDI Engines

	Particulate Emissions (mg/m^3)	
	IDI	DI
200 foot haul	111.6	112.0
700 foot haul	97.7	96.7

(Source: Reference 48)

From these findings, and others published by Mogan et al (84), it was found that particulate emissions varied considerably with changes in the F:A ratio. Interestingly, the ID and IDI engines both emitted similar levels of total particulate.

Using engines equipped with catalytic purifiers (84) it was shown that, at comparable F:A ratios, the IDI-Deutz-type engine generated 50% more combustible particulate before the converter than did a DI engine. At 1800 rpm, the particulate emissions decreased with increasing F:A ratio. Thus, it was suggested that less particulate would be emitted during a duty cycle if the engine speed did not exceed 1800 rpm.

Mogan and Stewart (85), in a paper on the relationship between CO and particulate emitted by surface vehicles, noted that for a similar diesel not derated for underground use, CO emissions would be about 5 times those of the derated engine or 3000 ppm CO, with particulate emissions of 500 mg/m^3. The derated version would emit about 600 ppm CO and 100 mg/m^3 of particulate.

In an extensive qualitative investigation which elaborated on earlier work (74, 76, 86), Mentser and Sharkey (87) explored the chemical composition of diesel particulate. Correlations were attempted for various operating modes for both an IDI and a DI engine and particulate size and composition. The type of engine or fuel used did not influence particulate composition greatly. Engine speed and load did, however, impact on the character of the particulate emissions. In the idle mode, the hydrocarbons contained both saturated and aromatics and terminated at C-20. This was similar to the composition of the fuel. As engine speed and load increased, there was an increase in the range of saturated aliphatics emitted up to about C-30. The range of saturated aliphatics were reduced at maximum load and rpm compared to those found during intermediate operating conditions.

Using mass spectrometry and computer matching techniques, it was found that diesel particulate contained straight-chain saturated hydrocarbons, aldehydes, ketones, esters and glycidols. In addition, a host of aromatics, including ethers, cyclic ketones and hydrides, alkylbenzenes, phenols, dihydric phenols, hydroaromatics and polynuclear aromatics (PNA's) were identified (87).

Because of concern over the carcinogenicity of PNA's, Mentser and Sharkey detailed their presence in diesel particulate (87). Table 32 lists PNA's with known carcinogenicity and their occurrence in diesel particulate (89). The $C_{18}H_{12}$ and $C_{20}H_{12}$ varieties were most prevalent.

Williams (88, 89) recently reviewed the composition, concentration and control of diesel particulate emissions and pointed out that no unique features of diesel particulate have yet been identified which clearly distinguishes it from the particulate of gasoline-burning engines or from particulate from other combustion sources. The per mile emissions of elemental carbon appears to depend more on the air-to-fuel ratio than on the fuel or engine type. Moreover, the BaP emissions from diesel vehicles does not differ relatively from those of gasoline-powered cars. However, diesel vehicles do emit larger amounts of particulate carbon on a per-mile basis than gasoline vehicles. It should be emphasized that much of the more recent data which has been published on diesel particulate, both in relation to its size and composition, has been largely a result of an increased impetus in determining the impact of the use of light diesel units in passenger cars and light-duty trucks.

TABLE 32 Frequency of Occurence of formulas for Carcinogenic Compounds in Diesel Exhaust Particulates and Respirable Coal Dust

Formula	Carcinogenic compound with corresponding formula	Carcinogenicity*	Molecular weight	Frequency of occurence in: Diesel exhaust particulates, 30 samples	Frequency of occurence in: Respirable mine dusts, 4 samples	Frequency of occurence in: Crushed coals, 4 samples
$C_{18}H_{12}$	Chrysene	±	228.0936	28	4	4
	Benzo(c)phenanthrene	+++				
	Benz(a)anthracene	+				
$C_{20}H_{12}$	Benzo(a)pyrene	+++	252.0936	16	4	3
	Benzo(b)fluoranthene	++				
	Benzo(j)fluoranthene	++				
$C_{20}H_{14}$	Benz(j)aceanthrylene	++	254.1092	2	3	--**
$C_{20}H_{16}$	7,12-Dimethylbenz(a)anthracene	++++	256.1248	1	3	1
$C_{21}H_{14}$	Dibenzo(a,g)fluorene	+	266.1092	2	3	1
$C_{20}H_{13}N$	Dibenzo(c,g)carbazole	+++	267.1045	1	--	--
$C_{21}H_{16}$	3-Methylcholanthrene	++++	268.1248	--	1	--
$C_{22}H_{12}$	Indeno(1,2,3-cd)pyrene	+	276.0936	3	3	1
$C_{22}H_{14}$	Dibenz(a,h)anthracene	+++	278.1092	2	--	--
	Dibenz(a,j)anthracene	+				
	Dibenz(a,c)anthracene	+				
$C_{21}H_{13}N$	Dibenz(a,h)acridine	++	279.1045	--	--	--
	Dibenz(a,j)acridine	++				
$C_{24}H_{14}$	Dibenzo(a,h)pyrene	+++	302.1092	--	--	--
	Dibenzo(a,i)pyrene	+++				
	Dibenzo(a,l)pyrene	+				

(*) The carcinogenicities are given in "Particulate Polycyclic Organic Matter", NAS, Washington, D.C. 1972 according to the following code: ± = uncertain or weakly carcinogenic; + = carcinogenic; ++, +++, ++++ = strongly carcinogenic

(**) Not detected

(Source: Reference 87)

Petersen (90) reported a refinement of an earlier fractionation method reported from Battelle-Columbus Laboratories which separates diesel particulate extract samples into six fractions including basics (Fraction 1), acids and phenols (Fraction 2), aliphatic hydrocarbons (Fraction 3), aromatic hydrocarbons (Fraction 4), moderately polar neutrals (Fraction 5) and highly polar neutrals (Fraction 6). The PAH compounds, nitrogen heterocycles and mononitrated PAH's are present in aromatic Fraction 4 with the polynitrated PAH sulphur heterocycles and oxygenated 2- and 3-ring PAH's being in the moderately polar Fraction 5. The highly polar Fraction 6 contains PAH with more than one functional group, oxygenated with more than 3 rings. The method is particularly useful as it can be readily applied to larger samples.

Funkenbusch, Leddy and Johnson (91) described their continuing investigations into the characterization of diesel particulate organic matter (POM). Their procedure begins with a Soxhlet extraction of the POM using dichloromethane to obtain the soluble organic fraction (SOF). The acidic and basic portions of the SOF are isolated by liquid-liquid extraction techniques using aqueous base and aqueous acid, respectively. The neutral portion of the extract is separated into paraffin, aromatic, transitional and oxygenated fractions by silica gel column chromatography. Two additional fractions, the ether and hexane insoluble fractions, are also separated by the procedure.

Quantitative mass data were determined on the fractionated extracts of 12 particulate samples from the exhaust of a medium-duty diesel engine collected in a dilution tunnel at an 8:1 dilution ratio. Three fuels with a range of properties and two steady-state engine conditions (Mode 3 and 5) from the EPA 13-mode cycle were used as experimental variables. The scheme for the extraction of SOF of diesel particulate is given in Figure 64. Table 33 provides an incomplete listing of organic compounds reported to be associated with POM. It should be noted that at 1700 rpm modes 3 and 5 correspond to 25 and 75% of full load, respectively. The data were tabulated with respect to the effect of engine mode and fuel type on the particulate extraction composition, as shown in Tables 34 and 35.

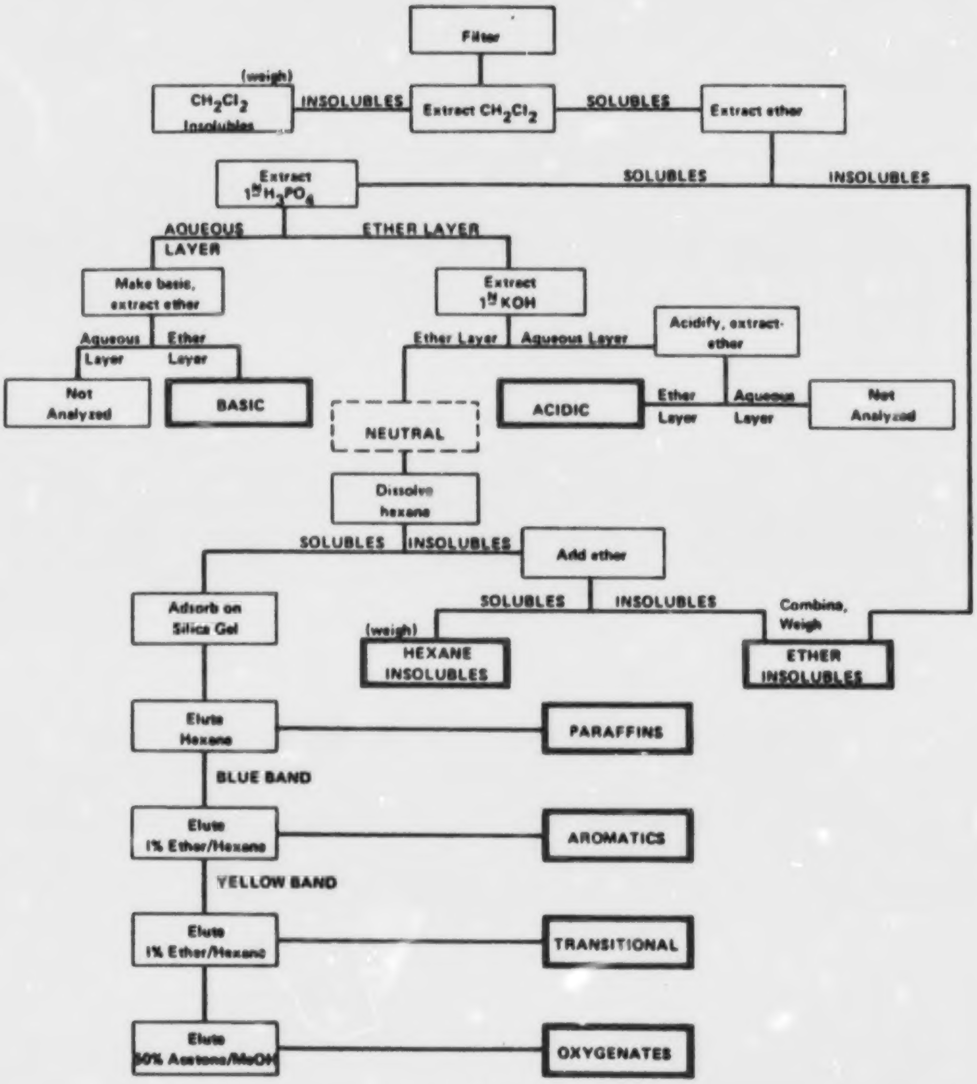

Filter
(weigh)
CH2Cl2 Insolubles
INSOLUBLES
Extract CH2Cl2
SOLUBLES
Extract ether
Extract 1N H3PO4
SOLUBLES
INSOLUBLES
AQUEOUS LAYER
ETHER LAYER
Make basic, extract ether
Extract 1N KOH
Acidify, extract-ether
Aqueous Layer
Ether Layer
Ether Layer
Aqueous Layer
Not Analyzed
BASIC
NEUTRAL
ACIDIC
Ether Layer
Aqueous Layer
Not Analyzed
Dissolve hexane
SOLUBLES
INSOLUBLES
Add ether
Adsorb on Silica Gel
SOLUBLES
INSOLUBLES
Combine, Weigh
(weigh)
HEXANE INSOLUBLES
ETHER INSOLUBLES
Elute Hexane
PARAFFINS
BLUE BAND
Elute 1% Ether/Hexane
AROMATICS
YELLOW BAND
Elute 1% Ether/Hexane
TRANSITIONAL
Elute 50% Acetone/MeOH
OXYGENATES

FIGURE 64

Scheme for Extraction of Soluble Organic Fraction of Diesel Particulates

(Source: Reference 91)

TABLE 33

Fuel Properties of Test Fuels

Acidic Compounds

Phenol
Cresol
Dinitro-o-cresol
Benzoic acid
o,m,p-Phenylphenols

Basic Compounds

Pyridine
Aniline
Benzacridene
o-toluidine

Paraffinic Compounds

Pentane
Hexane
Octane
n-Tetradecane
n-Pentadecane
n-Hexadecane
n-Octadecane
n-Nonadecane
n-Eicosane
n-Heneicosane
Cyclohexane
Methylcyclohexane

Aromatic Compounds

Benzene
Toluene
Diphenyl
Anthracene
Styrene
Xylene
Pyrene
Chrysene
Benzo(a)pyrene
Benzo(j)fluoranthene
Benzo(b)fluoranthene
Trimethylnaphthalene
Ethylfluorene

Transitional Compounds

Dioxane
Methoxyphenanthrene

Oxygenated Compounds

Hydroquinone
9,10-Anthraquinone
Furfural
Furfuryl alcohol
Cyclohexanol
2-Hexanone
Methylacetate
Crotonaldehyde
2-Pentanone

(Source: Reference 91)

TABLE 34

Comparison of Samples Based on Engine Mode

Sample	Fuel	% Extractable	% Paraffins	% Aromatics	% Transitional	% Oxygenated	% Acid	% Base	% Ether Insolubles	% Hexane Insolubles
Average of all Mode 3 Samples	A L L	81.2 ± 4.4**	57.6 ± 7.3	9.4 ± 3.1	4.6 ± 0.6	9.7 ± 3.0	6.8 ± 3.0	0.6 ± 0.5	10.4 ± 3.4	1.2 ± 0.6
Average of all Mode 5 Samples	A L L	21.7 ± 8.6	47.0 ± 10.2	3.9 ± 2.2	5.8* ± 0.7	11.3 ± 2.5	11.7 ± 2.5	1.4 ± 0.7	18.0 ± 7.8	1.7 ± 1.1
t-Test Confidence Level of Difference Between Modes		>99.9%	>90%	>99%	>99%	>95%	>95%	>95%	>90%	>80%

(*) The value for sample 20 - 32 was rejected by a Q-test at 90% confidence level.
(**) Standard deviation

(Source: Reference 91)

TABLE 35

Comparison of Samples Based on Test Fuel

Sample	Mode	% Extractable	% Paraffins	% Aromatics	% Transitional	% Oxygenated	% Acid	% Base	% Ether Insolubles	% Hexane Insolubles
Average of Fuel 1	ALL	48.2 ± 36.2*	48.8 ± 10.0	7.2 ± 5.3	4.6 ± 1.0	12.4 ± 2.2	9.1 ± 1.6	1.3 ± 0.9	15.6 ± 9.6	1.0 ± 0.9
Average of Fuel 2	ALL	47.7 ± 36.3	47.2 ± 8.0	7.0 ± 3.8	4.1 ± 2.5	11.4 ± 2.8	11.1 ± 5.3	0.8 ± 0.7	16.0 ± 5.2	2.4 ± 0.6
Average of Fuel 3	ALL	58.6 ± 31.1	61.1 ± 7.5	5.7 ± 3.2	5.4 ± 0.8	7.8 ± 0.9	7.6 ± 3.2	0.7 ± 0.6	11.0 ± 6.4	1.0 ± 0.0
t-Test Confidence Level of Difference Between Fuels 1-2		<80%	<80%	<80%	<80%	<80%	<80%	<80%	<80%	<99%
t-Test Confidence Level of Difference Between Fuels 1-3		<80%	<95%	<80%	<80%	<99%	<80%	<80%	<80%	<80%
t-Test Confidence Level of Difference Between Fuels 2-3		<80%	<95%	<80%	<80%	<95%	<80%	<80%	<80%	<99.9%

(*) Standard Deviation

(Source: Reference 91)

It was found that the SOF particulates generated during Mode 3 in general contained higher percentages of paraffin and aromatics and lower percentages of transitional, oxygenated, acid-base and ether-insoluble fractions in their organic solubles and particulate than those generated during Mode 5 operation. SOF particulates using fuel 3 contained higher percentages of oxygenated compounds than those from fuels 1 and 2 (see Table 36 for fuel properties). The authors emphasized that it was important to note that these compositional comparisons were on a relative, rather than an absolute, basis with respect to the total particulate mass in the exhaust. Figures 65 and 66 were provided to illustrate the effect of mode and fuel type on particulate emission concentrations. It was shown that the primary factors controlling the concentration of each fraction in the exhaust were the total mass concentration of particulates and the percentage of extractable materials in the particulates. These factors were highly dependent on the test variables of engine mode and fuel type. For a given engine mode, fuel 3 produced the highest concentration of particulates and fuel 1 the lowest. The percentage of extractable material was much higher for mode 3 than for mode 5 samples. Thus, for a given fuel, mode 3 engine operation produced higher exhaust concentrations of all of the organic soluble fractions than mode 5 engine operation; for a given engine mode, fuel 3 produced the highest exhaust concentrations of the soluble organic fractions and fuel 1 the lowest.

TABLE 35

Fuel Properties of Test Fuels

	Fuel 1	Fuel 2	Fuel 3
Gravity, °API	46.0	38.5	30.3
Viscosity, cSt, at 100°F	1.4	2.47	6.8
Cetane No. (D-976)	52.0	53.3	53.3
Pour Point, °F	-50	0	+50
Cloud Point, °F	-42	+4	+50
Carbon, Wt %	85.3	86.4	85.6
Hydrogen, Wt %	14.3	13.5	12.5
Oxygen, Wt %	0.01	0.022	0.21
Nitrogen, Wt %	0.0015	0.0051	0.14
Sulfur, Wt %	0.11	0.27	0.44
Distillation (D-86), %			
10	353	414	508
50	396	491	614
90	466	590	700
95	492	620	728
EP	518	655	743
Hydrocarbon Type by Mass Spectrometry, Wt % (C)			
Aromatics	15	21	27
Paraffins	51	40	27
Naphthenes	32	37	42
Sulfur Compounds	2	2	4
Hydrocarbons Type by FIAM (a)			
Aromatics	15	24	(b)
Paraffins and Naphthenes	85	76	(b)
Olefins	0	0	(b)

(a) The Fluorescent Indicator Adsorption Method (FIAM) is the ASTM D 1319 test procedure. This is a common method for measuring aromatics; however, it is designed primarily for gasoline and not for diesel fuel. The method specifies an upper limit of 600°F on end point of fuels tested.

(b) No separation was possible in this method.

(c) The 32 component hydrocarbon type analysis is based on the method in Reference 26. It utilizes high resolution mass spectrometry to distinguish between saturated and aromatic species that have the same nominal molecular weights but different accurate masses (e.g., eicosane, $C_{20}H_{42}$, M.W. 282.3286 and undecylnaphthalene, $C_{21}H_{30}$, MW = 282.2347) thus eliminating the need for physical separation of the species in order to measure their relative concentration.

(Source: Reference 91)

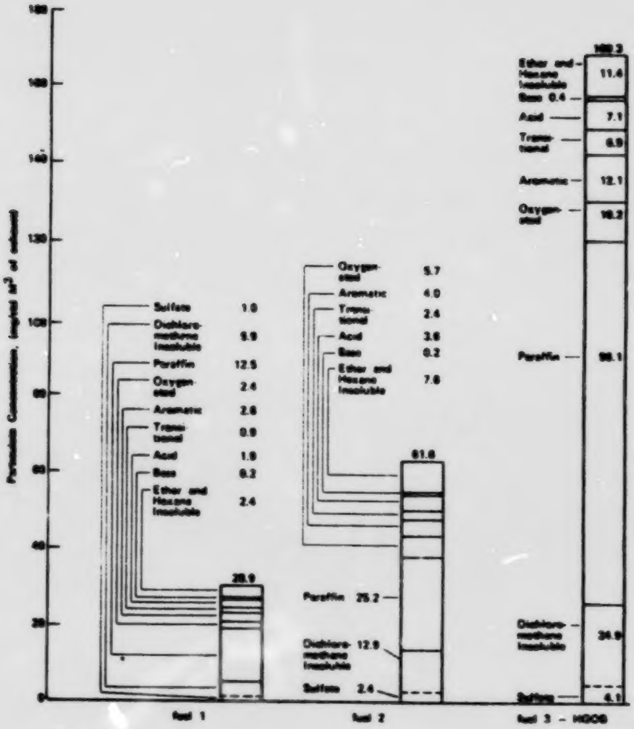


FIGURE 65

Fractional Breakdown by Mass Concentration of Soluble Organic Fraction for Mode 3 at 8:1 Volume Dilution Ratio

(Source: Reference 91)

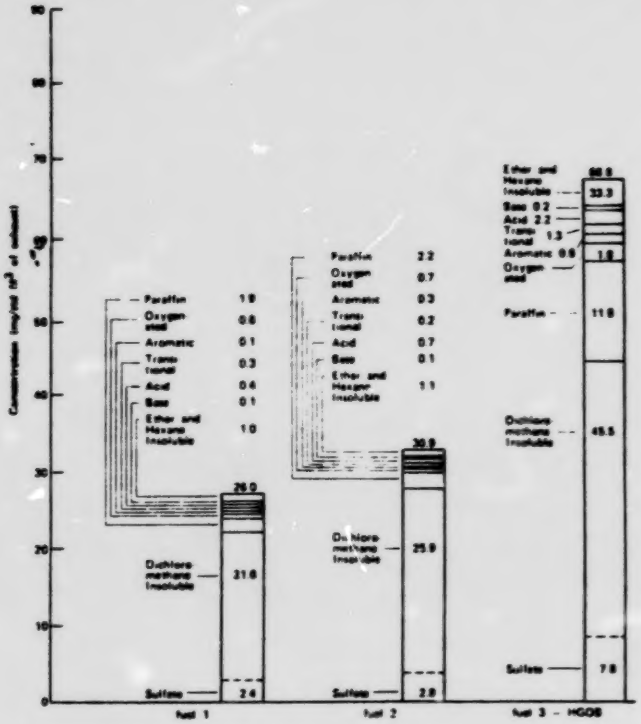


FIGURE 66

Fractional Breakdown by Mass Concentration of Soluble Organic Fraction for Mode 5 at 8:1 Volume Dilution Ratio

(Source: Reference 91)

Fractions of the SOF were characterized by several instrumental techniques and the following qualitative statements were made:

Paraffin (34 - 65% of SOF). Elemental, infrared and nuclear magnetic resonance analyses indicated that saturated hydrocarbon structures (alkanes) were by far the predominate species.

Aromatic (3 - 14% of SOF). Components of both aliphatic and aromatic character were indicated with no carbonyl or hydroxyl functionality. Alkyl substituted aromatic compounds were probably the major species present.

Transitional (less than 1 - 6% of SOF). Both aliphatic and aromatics were indicated and carbonyl functionality was clearly present. Aliphatic and aromatic ketones, aldehydes and esters were believed to be major constituents of this fraction.

Oxygenated (7 - 15% of SOF). Aliphatic and aromatic compounds with both carbonyl and hydroxyl functionality were present. This fraction probably contained a wide variety of higher polar compounds, including aromatic alcohols not acidic enough to be extracted in the acid-base separation.

Acidic (3 - 15% of SOF). Both aliphatic and aromatic types were indicated. A great variety of aliphatic and aromatic carboxylic acids and phenols were believed to be present.

Basic (less than 1 - 2% of SOF). This fraction was not studied because of its small mass. Other workers have identified azaheterocyclic polyaromatics in the base fraction of airborne particulate extracts.

Ether Insolubles (6 - 25% of SOF). Both aromatic and aliphatic components with a surprising amount of functionality, including hydroxyl and carbonyl, was indicated. This fraction could conceivably contain inorganic compounds, as well as organic compounds. The organics might be expected to be high molecular weight species of limited solubility. In some samples, particularly Mode 5 samples, glass fibers from the filter may have contributed substantially to this fraction.

Hexane Insolubles (1 - 3% of SOF). Limited investigation was done on this fraction because of its small mass. The samples were solid materials with an infrared spectrum nearly identical to that of the ether insoluble fraction.

In order to avoid the artifacts that develop in the collection process associated with filter sampling, Chan et al (92) used electrostatic precipitation (ESP) in parallel with filter sampling to examine the role of collection methods on the chemical and biological properties of diesel exhaust particulate. Electrostatic collection of particles results in a load pressure drop across the collector and allows the exhaust gases to pass over the collected particles, instead of through them. As a result, loss of the volatile compounds due to the partial vacuum and the mass transfer to particulate of gaseous hydrocarbons and nitrogen oxides should be reduced since only a small fraction of the gasses are in contact with the diesel particulate.

The extraction of diesel particulate was carried out using dichloromethane (DCM) in a Soxhlet apparatus.

The biological activities of the extracts were investigated and studied using the short-term Ames test. Typically, the mutagenic activity recovered from all the fractions represented between 75 -90% of the original extract. Considerably more detail on the mutagenicity of diesel particulate may be found in Section 4.5.2.

The collection efficiency of the diesel particles was 92% using the electrostatic sampler.

Chemical profiles of the particulate extracts indicated that more than 60% of the extractables were in the neutral-nonpolar I fraction (Figure 67).

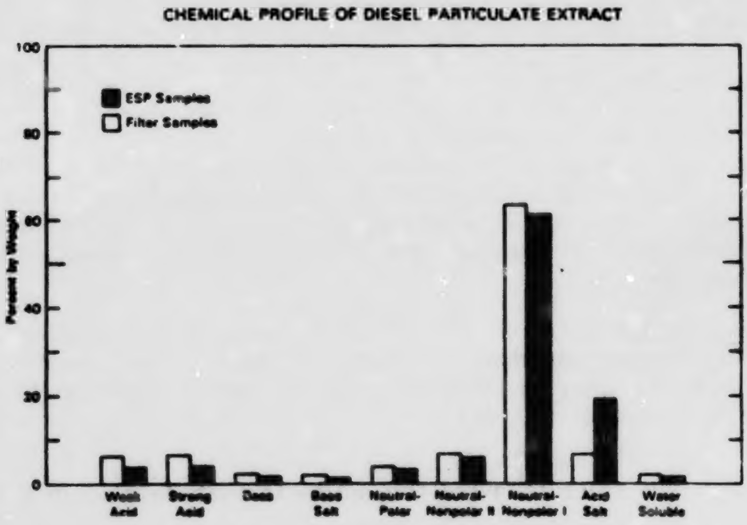


FIGURE 67

Chemical Fraction of Diesel Particulate Extracts for both ESP and Filter Samples. The NEUTRAL-NONPOLAR I Fraction accounted for more than 60% of the Extractables

Source: Reference 92

It was found that the amount of extractable compounds from diesel exhaust depended on the collection temperature and method. At a given collection temperature more extractables were found in diesel particles collected by ESP than by filtration. Chemical profiles of extracts of both ESP and filter collected particulate were similar with over 60% of the extractable mass classified as neutral-nonpolar I fraction. This fraction was obtained using a solvent partitioning scheme and probably contained aliphatic and 1-ring aromatic hydrocarbons.

Most of the mass increase in the extractables of the ESP sample was found in an acid-salt fraction and was attributed to charging effects. Although there was more than a two-fold increase in the extractables in this fraction, no bioactivity was observed in the Ames test.

Bioactive compounds in the Ames test were found in the neutral-polar and nonpolar II fractions. These fractions accounted for more than 70% and 90% of the bioactivity in the filter and ESP samples, respectively. However, only 10 - 15% by weight of the extractables belonged to these fractions.

Of considerable interest and importance was the finding that respirable coal dust also contained some PNA compounds. However, PNA's associated with coal dust sampled from a section of a mine using diesel equipment had qualities which resembled that seen for diesel particulate alone. Although the importance of this study is recognized, it is unfortunate that the techniques used provided only qualitative information and did not permit quantitative determinations to be made.

As will be discussed in more detail in Section 3.4.8, wet scrubbers result in modest reductions in diesel particulates. Catalytic converters alone do not materially influence particulate output. When a wet scrubber is used in tandem with a platinum exchanger, a synergistic effect is obtained which can lead to particulate reductions in the range of 44 - 55% (93).

3.4.8 Polynuclear Aromatic Hydrocarbons

In Sections 3.3.1 and 3.4.7, it was pointed out that many chemical substances are emitted as a result of the incomplete combustion of diesel fuel. One particular "family" of compounds includes the potentially carcinogenic polynuclear aromatic hydrocarbons which are variously referred to as PNA's or PAH's (polycyclic aromatic hydrocarbons). These substances have been identified in both automobile (12) and diesel exhausts (87) and were recorded in the literature of the 1950's (86) and 1960's (94).

Because some research groups attempted to correlate the fractionation and identification of the PAH's attached to diesel particulate with pulmonary deposition and mutagenic activity, some of the exhaust constituent data overlaps with several other sections, including 4.5.1, 4.5.2 and 4.5.3 and should be reviewed in conjunction with them.

Unfortunately, even though PNA's have been recognized as being present in diesel exhaust, the quantitative aspects of their presence has remained unclear. This results largely from the fact that some of the compounds are unstable and have a tendency to "stick" onto glass, plastic and particulate matter, thus making accurate sampling difficult. Further, it is only in the past 5 - 10 years that techniques, such as gas-liquid chromatography (GLC), high pressure liquid chromatography (HPLC), mass spectrometry (MS) and combinations of these instrumental techniques, have become available which provide sufficient analytical resolution to separate members of the family with reasonable quantitative sensitivity. Extremely sensitive techniques are required, as PNA's are present in exhaust in the order of nanograms (ng) per m^3. To accentuate these difficulties is the large number of "relatives" in the "family" that must be differentiated and quantitated. Some members are non-carcinogenic, at least as determined by animal testing, whereas others are potent carcinogens (95).

A list of polycyclic compounds, their occurrence in diesel exhaust and their relative carcinogenicity is given in Table 37.

TABLE 37

Occurrence of Carcinogenic Compounds in Diesel Exhaust and Their Relative Carcinogenicity

Formula	Carcinogenic compound with corresponding formula	Carcinogenicity*
$C_{18}H_{12}$	Chrysene	±
	Benzo(c)phenanthrene	+++
	Benzo(a)anthracene	+
$C_{20}H_{12}$	Benzo(a)pyrene	+++
	Benzo(b)fluoranthene	++
	Benzo(j)fluoranthene	++
$C_{20}H_{14}$	Benz(j)aceanthrylene	++
$C_{20}H_{16}$	7,12-Dimethylbenz(a)anthracene	++++
$C_{21}H_{14}$	Dibenzo(a,g)fluorene	+
$C_{20}H_{13}N$	Dibenzo(c,g)carbazole	+++
$C_{21}H_{16}$	3-Methylcholanthrene	++++
$C_{22}H_{12}$	Indeno(1,2,3-cd)pyrene	+
$C_{22}H_{14}$	Dibenz(a,h)anthracene	+++
	Dibenz(a,j)anthracene	+
	Dibenz(a,c)anthracene	+
$C_{21}H_{13}N$	Dibenz(a,h)acridine	++
	Dibenz(a,j)acridine	++
$C_{24}H_{14}$	Dibenzo(a,h)pyrene	+++
	Dibenzo(a,i)pyrene	+++
	Dibenzo(a,l)pyrene	+

(*) The carcinogenicities are given in "Particulate Polycyclic Organic Matter", NAS, Washington, D.C. 1972, according to the following code:

± = uncertain or weakly carcinogenic
+ = carcinogenic
++, +++, ++++ = strongly carcinogenic

(Source: Reference 87)

The chemical structure of several PNA's associated with diesel exhaust have been reproduced in Figure 68.

At the 1977 - NIOSH Diesel Workshop, the concensus of opinion was that current analytical methodology was inadequate to permit articulation of even a qualitative emission factor for PNA's. However, individuals who were actively engaged in PNA analyses, voiced the opinion that the group were too conservative and that methodology was sufficiently sensitive to permit reliable estimates to be made. In the succeeding period methodology has become sufficiently reliable, both qualitative and quantitatively, to obviate the need to criticize the value of the data published in the past 3 - 5 years.

Even methods, such as those used by Spindt (96, 97), in which recovery of at least a few selected PNA's are determined using a radioactive ^{14}C tracer technique and gas chromatography-mass spectrometry (98) had credibility at the time of issuing the original version of this review.

Spindt (97) reported the minimum detectable limits for 11 PNA's in diesel fuel shown in Table 38.

TABLE 38

Minimum Detectable Limits of PNA's As Reported by Spindt

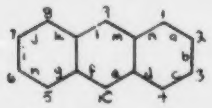
	Limit of Detection
Benzo(a)pyrene (BaP)	0.5 ug/kg (ppb)
Benzo(e)pyrene (BeP)	1.0 ug/kg (ppb)
Benz(a)anthracene (BaA)	1.0 ug/kg (ppb)
Benzo(ghi)perylene (BghiP)	0.5 ug/kg (ppb)
Chrysene (Chry)	1.0 ug/kg (ppb)
Tribenzo(h,rst)pentaphene (Tri)	1.5 ug/kg (ppb)
Pyrene (P)	5.0 ug/kg (ppb)
Fluoranthene (Fluo or Ft)	5.0 ug/kg (ppb)
Anthracene (A)	5.0 ug/kg (ppb)
Phenanthrene (Phen)	5.0 ug/kg (ppb)
Methylanthracene (M-Anth)	5.0 ug/kg (ppb)

(Source: Reference 97)

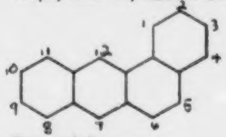
FIGURE 68

The chemical structure of some PNA's associated with diesel exhaust (Source: Reference 27)

Anthracene, $C_{14}H_{10}$
m.p., 216°C; b.p., 340°C


8
9
1
7
2
3
6
5
10
4

Benz(a)anthracene, $C_{18}H_{12}$
m.p., 158°C; sublimes (1,2-benzanthracene)


2
1
3
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11
12
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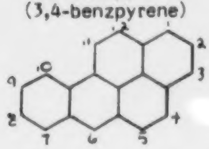
8
7
6
5

Benzo(a)pyrene
(1,2-benzpyrene)
(3,4-benzpyrene)


12
1
11
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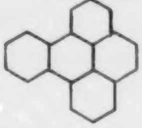
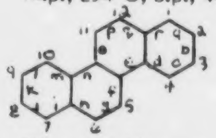
4
7
6
5

Benzo(e)pyrene
(4,5-benzpyrene)
(1,2-benzpyrene)



Chrysene
= 1,2-benzophenanthrene
$C_{18}H_{12}$
m.p., 254°C; b.p., 448°C


12
11
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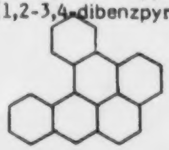
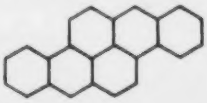
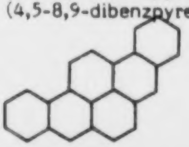
Dibenzo(a,l)pyrene
(2,3-4,5-dibenzpyrene)
(1,2-3,4-dibenzpyrene)



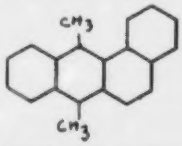
Dibenzo(a,h)pyrene
(1,2-6,7-dibenzpyrene)
(3,4-8,9-dibenzpyrene)



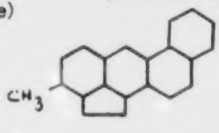
Dibenzo(a,i)pyrene
(2,3-6,7-dibenzpyrene)
(4,5-8,9-dibenzpyrene)



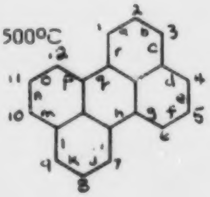
7,12-Dimethylbenz(a)anthracene
(9,10-dimethyl-1,2-benzanthracene)



3-Methylcholanthrene



Perylene, $C_{20}H_{12}$
m.p., 273°C; b.p., ca. 500°C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8

In the first report of a study on the PNA content of heavy-duty diesel exhaust carried out at Gulf Research for the Coordinating Research Council, Spindt (97) published values for the PNA content of 5 different samples of Number 1, and 17 samples of Number 2 diesel fuels. A selection of values from this report are given in Table 39. The data clearly show that there is a great deal of variability in the PNA content of fuels, particularly for No. 2 fuel. The other, possibly more important point which emerged, was the generally, and significantly, lower PNA content of No. 1 fuels, compared to No. 2 fuels.

TABLE 39

PNA Content of Diesel Fuels (ug/kg)*

(No. 2 Diesel Fuels)

	Compound						
Code No.	BaP	BeP	BaA	Chry	Fluo	Anth	M-Anth
2-1	69	177	132	452	568	2,868	9,300
2-6	274	52	422	105	3,721	11,800	69,800
2-7	86	63	364	322	1,454	19,700	43,200
2-10	2	1	1	ND	ND	2000	ND
2-16	41	75	108	72	869	31,400	19,700

* Equal to parts per billion (ppb)

ND = not detected

(Source: Reference 10)

Using a blend of No. 2 fuels, indicated as Code No. 2-16 in Table 39, the emission of PNA's in diesel exhaust was evaluated using an automotive diesel engine and the EPA 13-mode cycle. In Table 40, the emissions of specific PNA's are expressed related to exhaust gas, fuel burned and fuel content. To permit those interested to relate these values to engine operating parameters and other exhaust emissions, Table 41 is provided.

Of significance was the finding that the amount of PNA in the exhaust was much greater than the 0.5% expected, had the exhaust PNA come only from the fuel (Table 42). This led the authors to

TABLE 40

Average PNA Emissions From Four 13-Mode cycles

DDAD 3-53 Engine - Fuel 2-16

Compound	ug/m^3 Exhaust Gas	ug/gal Fuel Burned	ug/kg Fuel Burned	ug/kg In Fuel
BaP	0.158	22	6.9	4.1
BeP	0.031	4	1.3	75
BaA	--	--	--	108
BghiP	0.048	7	2.2	26
Chrysene	0.035	5	1.6	72
Pyrene	2.501	349	110	144
Anthracene	--	--	--	31,400
Fluoranthene	1.88	253	79	869
Phenanthrene	46.1	6,410	2,013	--
Phenanthrene Der.	59.8	8,280	2,600	--
Phenols	107.2	14,900	4,680	35,000

(Source: Reference 45)

TABLE 41

Gaseous Emission Summary - 13-Mode Cycle

(DDAD 3-53 Engine - Fuel 2-16)

						Modal Data									
							Concentration,ppm				Mass, g/hr				
Mode	rpm	Hp	% Load	Bsfc lb/ bhp-hr	Exh. Flow lb/hr	Fuel/Air[1] Ratio	CO	NO_2	NO	HC	CO	NO_2	NO[2]	HC	Smoke, Percent Opacity
1	505	0.3	--	4.597	241	0.0054	263	28	139	190	27.6	4.5	21.9	10.0	0.6
2	1816	2.7	4.0	2.497	833	0.0083	325	29	128	271	116.8	15.6	68.8	50.2	0.5
3	1822	16.8	24.7	0.606	837	0.0123	238	45	222	240	85.1	24.1	118.6	44.7	0.6
4	1820	33.9	50.0	0.455	845	0.0186	213	80	407	238	76.1	43.0	218.0	44.9	0.8
5	1813	50.7	74.7	0.433	843	0.0268	400	149	633	250	140.6	78.8	335.7	47.0	2.5
6	1817	67.9	100.0	0.450	845	0.0375	5788	206	857	140	1992.1	108.6	448.2	26.7	14.2
7	500	0.2	--	6.180	240	0.0053	188	30	146	157	19.5	4.7	22.5	8.6	0.6
8	2807	86.9	100.0	0.466	1222	0.0343	1150	269	843	275	587.5	204.4	642.0	75.3	4.2
9	2797	65.0	74.8	0.484	1214	0.0266	250	165	618	275	126.7	125.6	470.6	74.5	2.0
10	2801	43.7	50.3	0.558	1206	0.0206	175	65	382	265	89.1	49.8	291.4	71.7	1.3
11	2795	21.9	25.2	0.815	1205	0.0150	175	47	230	266	89.9	35.8	176.4	72.0	1.0
12	2802	3.2	3.7	4.862	1209	0.0130	175	41	181	279	90.4	31.5	140.0	75.8	0.8
13	505	0.3	--	4.203	239	0.0050	188	34	136	190	19.5	5.2	20.8	10.3	0.6

13-Mode cycle Average, g/bhp-hr[3]					
Bsfc	bhp	CO	NO_2	NO[2]	HC
0.5472	31.48	8.82	1.85	7.53	1.54

(1) Includes Scavenger Air

(2) Calculated as NO_2

(3) Calculated as Specified in Federal Register

(Source: Reference 45)

TABLE 41

Comparison of Exhaust PNA to PNA in Fuel

	Fuel 2-5		Fuel 2-16	
	ug/kg Fuel Burned	% of Fuel PNA in Exhaust	ug/kg Fuel Burned	% of Fuel PNA in Exhaust
BaP	2.7	30	7	17
BeP	3.3	32	1	2
BaA	2.7	18	--	0
BghiP	6.5	72	2	8
Pyr	121	27	110	76
Chry	8.2	1	3	4
Anth	0	0	0	0
Flou	0	0	85	10
Phen	2,409		2,015	
Phen Der.	--	--	2,396	
Phenols	5,600	15	4,723	13

(Source: Reference 45)

conclude that the excess of the PNA's in the diesel exhaust was a result of incomplete fuel combustion. Whether this data, derived using an automobile engine and No. 2 fuel, was reliable and relevant to heavy-duty diesel engines was not known until recently when Spindt (96) published his second annual report on the PNA content of exhaust from a heavy-duty diesel engine.

In the more recent study (96), Spindt pointed out that the previous findings were not reproducible and improved sampling procedures were developed for the heavy-duty engine experiments.

In the latter study, the emission of BaP and BaA from a Mack ENDT-675 engine were measured. Data were reported as shown in Table 43.

TABLE 43

PNA Emissions From a Heavy Duty Diesel Engine

	ug/kg/Fuel		ug/m^3 Exhaust	
	BaP	**BaA**	**BaP**	**BaA**
No load, 1260 rpm	25	68	0.4	1.0
50% load, 1230 rpm	8	32	0.3	1.1
100% load, 1800 rpm	16	16	0.6	0.7

(Source: Reference 96)

A comparison of these findings with those in Table 40 show that the PNA output from the heavy-duty engine was greater than from the lighter automobile diesel. The authors noted that in the earlier investigations, when the exhaust cooled, the PNA's condensed and deposited on the surfaces of the lines and filters leading to the trap, resulting in poor reproducibility and low recoveries. This was overcome in the more recent work by using an improved trapping system.

Chaudry & Busch (15) considered computerized gas chromatography-mass spectrometry (GC-MS) to be most powerful technique available for the identification of PAH's and other trace organics in a complex environmental sample.

A two-step clean-up procedure was found to be highly effective for the isolation of PAH. No interfering species were detected by mass spectrometry. The compounds were primarily identified by retention times using high resolution GC and mass spectra of the GC-separated components.

The GC profile of the PAH fraction from a VW Rabbit exhaust sample is shown in Figure 69. Comparison of the peak retention times with those representative of parent PAH's indicated that the major components were low molecular weight PAH's. The minor components could not be identified with a high degree of confidence. Four parent

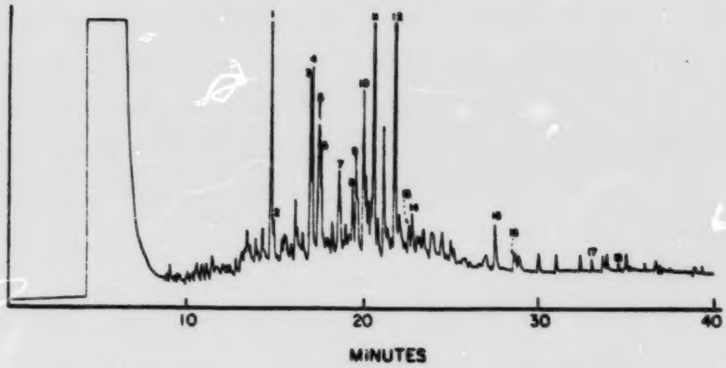


FIGURE 69

Gas Chromatogram of PAH fraction of diesel particulate extract (VW Rabbit). GC condition: 45-m x 0.35-mm id glass capillary column coated with SE54. Flame ionization detector. Temp: 110° for 2 min, programmed to 170° at 10°/min., to 212° at 3°/min., to 278° at 7°/min., to 290° at 8°/min.

Peak Identities:

1.	Phenanthrene
2.	Anthracene
3-6.	Methylanthracene/phenanthrene
7.	2-Phenylnaphthalene
8-10.	Dimethylanthracene/phenanthrene
11.	Fluoranthene
12.	Pyrene
13-14.	Trimethylanthracene/phenanthrene
15.	Benzo(ghi)fluoranthene
16.	Chrysene Benzo(a)anthracene Triphenylene
17.	Benzofluoranthene
18.	Benzo(e)pyrene/benzo(a)pyrene

(Source: Reference 15)

PAH's were identified, including phenanthrene (peak 1), anthracene (peak 2), fluoranthene (peak 11) and pyrene (peak 12).

Additional minor components, characterized tentatively by mass spectrometry, were isomeric methyldibenzothiophenes/dibenzodioxanes. Other isomeric compounds, such as benzo(a)fluorene and benzo(b)fluorene, were also present.

Since mass spectrometry alone cannot differentiate between isomeric PAH's, additional independent evidence for identification of individual isomers must be found. This is particularly important because the carcinogenic properties of PAH's may be dependent on isomeric structure. PAH's absorb strongly in the ultraviolet region and advantage was taken of this characteristic to achieve unambiguous characterization. High pressure liquid chromatography (HPLC) retention times could also be used for identification, as HPLC gives excellent separation of PAH's.

The GC-MS profile of the PAH fraction of a particulate extract from a Mercedes 300D was very similar to that from the VW Rabbit. The chromatogram seen in Figure 70 shows the presence of phenanthrene, anthracene, methylphenanthrene and other compounds including chrysene, all of which have cancerogenic potential.

Perez (99) also reported on efforts to chemically characterize diesel exhaust components from 3 heavy duty engines (Table 44). Some 9 carbonyl compounds were found but no cyanides were detected. The minimal detectable concentration of H_2S was 0.1 ppm, showing the low level of hydrogen sulphide in these exhausts. Most metals, with the exception of traces of zinc and calcium, were not detectable. The presence of dimethylnitrosamine (DMNA) and dibutylnitrosamines (DBNA) could not be verified using GC-MS techniques.

A great deal of effort was made to examine the particulate fraction of exhaust with 14 PNA's being identified (Table 45). Sulphuric acid aerosols and other sulphates in diesel exhaust were collected on fluorocarbon membrane filters. The concentration of sulphur dioxide in diesel exhaust was measured as sulphate using ion chromatography.

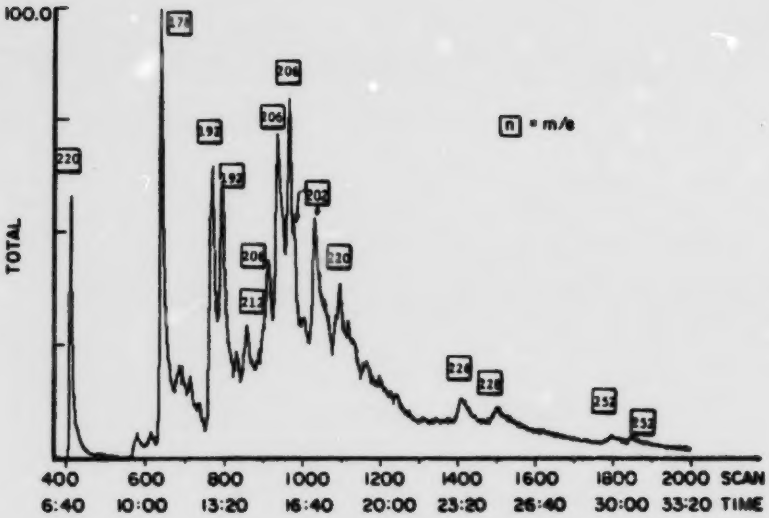


FIGURE 70

Total Ion Chromatogram of PAH Fraction of Diesel Particulate Extract (Mercedes 300-D)

(Source: Reference 15)

TABLE 44

Heavy Duty Diesel Engines

Engine	A	B	C
Type	DINA	DI-EGR	DITA
No. Cylinders	8	8	6
Bore & Stroke, mm	114 x 127	114 x 127	137 x 165
Rated Speed, RPM	2800	2800	2100
Rated Power, Kw	157	149	284
FEDERAL TEST CYCLE DATA, G/BHP-HR:			
CO	5.92	5.37	2.20
HC	0.77	0.57	0.27
HC + NO_2	8.89	5.73	7.93

DI = Direct Injection
NA = Naturally Aspirated
EGR = Exhaust Gas Recirculation
T = Turbocharged
A = Aftercooled

(Source: Reference 99)

TABLE 44

Relative Retention Times of Various PNA Relative to Fluoranthene

PNA	Retention Ratio
Acenaphthene - (Acn)	0.70
Phenanthrene - (Ph)	0.74
Anthracene - (An)	0.79
Fluoranthene - (Fla)	1.00
Pyrene - (Py)	1.14
Benz(a)anthracene - (BaA)	1.39
Chrysene - (Chr)	1.38
Benz(e)pyrene - (BeP)	1.96
Perylene - (Per)	1.96
Benzo(k)fluoranthene - (BkF)	1.96
Benzo(a)pyrene - (BaP)	2.23
Dibenz(a,h)anthracene - (DBahA)	2.52
Benzo(g,h,i)perylene - (BghiP)	3.25
Dibenz(a,i)pyrene - (DBaiP)	4.96

(Source: Reference 99)

In attempting the measurement and chemical characterization of the solvent extractable fraction of exhaust, which contains most of the PNA's, a special fractionation system was employed. Chemical separation and HPLC were used to characterize the soluble extractable fractions (SEF's). The percent of aromatic hydrocarbons, polar and oxy-compounds were determined using the system described in the article.

Sulphur dioxide and SO_4 emission values shown in Table 46 were consistent with fuel consumption and accounted for about 90% of the fuel sulphur.

Ammonia, although not expected to be in diesel exhaust, was detected at levels of 0.7 -1.15 mg/BHP-hour. Background levels or

TABLE 46

Sulphur Compounds

Engine	SO_2 g/BHP-hr	SO_4 mg/BHP-hr	H_2S ppm
A	0.88	8.08	<0.1
B	1.01	10.2	<0.1
C	0.91	17.2	<0.1

(Source: Reference 99)

interferences with the method produced positive results of about the same magnitude. The maximum corrected concentration found in the exhaust was 0.4 mg/m^3 and, whether real or not, is well below the recommended OSHA levels of 35 mg/m^3.

The analytical procedure employed has the potential for detecting a number of PNA's, but to date most of the work has involved BaP and, to a lesser extent, BaA (benzanthracene). The GC-MS method has been used to verify BaP and BaA values. The composite of cyclic-BaP values in micrograms per BHP-hour for 3 engines is shown in Table 47 and are consistent with previous reports. However, fuel BaP values appear to be consistently higher than those reported in a survey of 20 diesel fuels. Although engines were efficient in burning the fuel, some of the exhaust BaP could have originated from the fuel as indicated by the ratios in Table 48. It was noted that Johnson and co-workers had recently reported that hydrocarbons can be absorbed onto the surface of particulates and that hydrocarbon aerosols can readily form during cooling. The inability to obtain a hydrocarbon material balance in the Perez experiments suggested a significant unaccounted fraction. The work raised questions regarding the extensive amount of risk assessment work underway using methylene chloride extractable particulate fractions. Perez questioned whether the right materials were being tested. He also asked whether some of the differences seen between laboratories in mutagenic and animal tests were due to differences in particulate collection methods.

TABLE 47
BaP by HPLC

Engine	ug/BHP-hr	Range, mg/Kg Fuel	Fuel BaP, mg/Kg Fuel
A	1.08	0.001 - 0.031	4.7
B	4.34	0.001 - 0.199	14.6
C	0.34	0.001 - 0.021	12.7

(Source: Reference 99)

TABLE 48
Exhaust:Fuel BaP

	ug BaP/BHP-hr	mg BaP/Kg Fuel	Ratio*
Engine A	1.08	4.7	0.0012
Engine B	4.34	14.6	0.0014

$$*\text{Ratio} = \frac{\text{BaP Out in Exhaust}}{\text{BaP In Fuel In}}$$

(Source: Reference 17)

Schuetzle and co-workers (100, 101) used a multitude of analytical approaches to analyze for nitrated polycyclic aromatic hydrocarbons in light engine diesel exhaust particulate. They used a combination of high-performance liquid chromatography, GLC/MS, high-resolution MS and MS/MS techniques. Much of the work was directed at identifying the nitrated-PAH's as it was thought that they could make a significant contribution to the mutagenicity of diesel particulate. In particular, 1-nitropyrene was identified and it was calculated that it alone could account for up to 30% of the TA-98 direct-acting mutagenicity of unfractionated exhaust extracts. Details of the biological portions of their work can be found in Section 4.5.2.

In keeping with data mentioned earlier, and discussed in more detail in Section 4.5.2, Henderson et al (102) found that NO_2 treatment of aromatic and aliphatic components of diesel exhaust significantly increased their cytotoxicity and mutagenicity.

Eisenberg, Gordon and Perez (103) emphasized the possibility that some polycyclic aromatic hydrocarbons may not be isolated when using particulate filters during collection of heavy-duty exhaust emissions. They identified over 40 compounds in the gas phase after particulate filtration of which 35 were found to be volatile PAH's. The authors emphasized that since the role of these compounds in the formation of artifacts in mutagens was not resolved, the methods for measuring PAH in exhaust streams needed to be modified to account for the gas-phase compounds.

Perez (99) concluded that in comparison to established OSHA and ACGIH values, none of the substances measured were present in exhaust at concentrations that could be considered dangerous, at least for short-term exposure periods. He considered the immediate concern to be to establish the significance of the material passing through the particulate filter into the gaseous phase.

Additional publications, of relatively minor interest, have described advances in the analyses of PAH's. For example, Jentoft and Gouw (104) described the use of supercritical fluid chromatography, with CO_2 as the mobile phase, for the isolation of PAH's, such as BaP and benzanthracene, from a matrix of predominantly PAH's. This method can be used to separate various PAH's from each other with the lower detection limit being a few ppb in a liquid sample.

Nielsen (105) reported on a high-performance liquid chromatographic (HPLC) method with fluorescent detection for the determination of polycyclic aromatic hydrocarbons in particulate matter in automobile exhaust and in fuels and oils. It was thought that the fluorescent detector, being generally more sensitive, would result in the need for smaller samples and give better quantitative data and that the HPLC approach would be less time-consuming and more problem-free than using GLC. Although not directly relevant to diesel exhaust, the retention time and sensitivity data for various PAH's suggested that this could be a useful technique in the routine analysis of PAH's.

Sawicki (106) published GLC and TLC techniques with UV detection for the analysis of polynuclear aromatic hydrocarbons in automobile exhaust.

Tejada (107) developed a reverse-phase HPLC-fluorescence technique for the detection, identification and measurement of selected nitro-aromatic compounds with high sensitivity at the sub-nanogram level in various diesel exhaust particulate extracts.

Wotzak and Whitby (108) reported on the utility of a relatively new technique, total luminescence spectroscopy, in the analysis of organic extracts of diesel particulate matter.

Other laboratories have published information on the use of mass spectrometry for the direct characterization of diesel particulate matter (109).

Williams and Chock (110) summarized the physical and chemical characteristics of diesel exhaust with an emphasis on light-duty motors which is not necessarily typical of underground load-haul-dump equipment. Of interest in their report was that the BaP content of exhaust tended to increase with dilution, which was interpreted as indicating that sampling in a concentrated exhaust stream caused significant BaP to be converted to other products, possibly by reaction with NO_2. Their experiments strongly suggested that the hydrocarbons were bound to the particles as they left the tailpipe and that the hydrocarbons remained attached, at least through the initial dilution process.

Shabad (111), in a review of the potential carcinogenicity of automobile gasoline exhaust, noted that others had reported that soot scraped off the internal surface of exhaust tubes of cars contained 200 mcg/g of BaP, whereas diesel engines only contained 1 mcg/g of BaP. Further, studies showed that skin application of benzene extracts from carburator soot induced a higher incidence of papillomas and carcinomas in mice, whereas extracts from diesel soot did not. In discussing the hazard for man of pollution due to road transport vehicles, it was noted that the mean 24-hour concentration of BaP in the air near large highways had been reported to be as high as 3 mcg/100 m^3. It was calculated that a man inhaling 20 m^3 of air daily would receive a daily average of nearly 0.6 mcg of BaP due to automobile exhaust, an amount equal to that in the smoke of 50 cigarettes. They also reported on investigations on the BaP content of aviation engines and found that each turbo-jet or turbo-prop engine of a modern plane was capable of discharging 2 - 4 mg BaP per minute into the atmosphere. Thus, at a modern airport an annual discharge of up to 2 kg per year of BaP could easily occur. The amount of PAH, and especially BaP, discharged into a car park of a big city could be considerable with one car discharging up to 1 kg per year of BaP into the atmosphere, which lead Shabad to conclude that road transport was the main source of atmospheric pollution by PAH.

Yergey, Risby and Lestz (112) summarized their work on the chemical characterization of diesel particulate at a recent symposium. Their research was aimed at understanding the mechanism involved in the formation of particle-adsorbed species. HPLC data showed that unsubstituted, non-linear, polynuclear aromatic compounds were the primarily particle-bound combustion products from aliphatic hydrocarbon fuel components. Nitropyrene, a known carcinogen, was tentatively identified in the soluble organic fraction of the particles and it was suggested that it could account for a large portion of the observed mutagenic property of diesel exhaust organic extracts.

For comparative purposes, the work of Grimmer and co-workers (113, 114, 115 and 116) describing the sampling and analytical determination of the PAH content of gasoline powered automobile exhaust will be briefly discussed.

Grimmer and Hildebrandt (114) measured the PAH emission from a group of cars undergoing the EUROPA-Test cycle, which simulates city driving, and found that the total amount of 14 selected PAH's emitted by all test vehicles during the test cycle was in the range of 1 - 16 mg. It was found that although the absolute amount of a single PAH in the exhaust varied from car to car, the relative amounts appeared in a definite ratio. Figure 71 shows the relationship of the 14 PAH's from the 100 vehicles investigated, with average values and ranges.

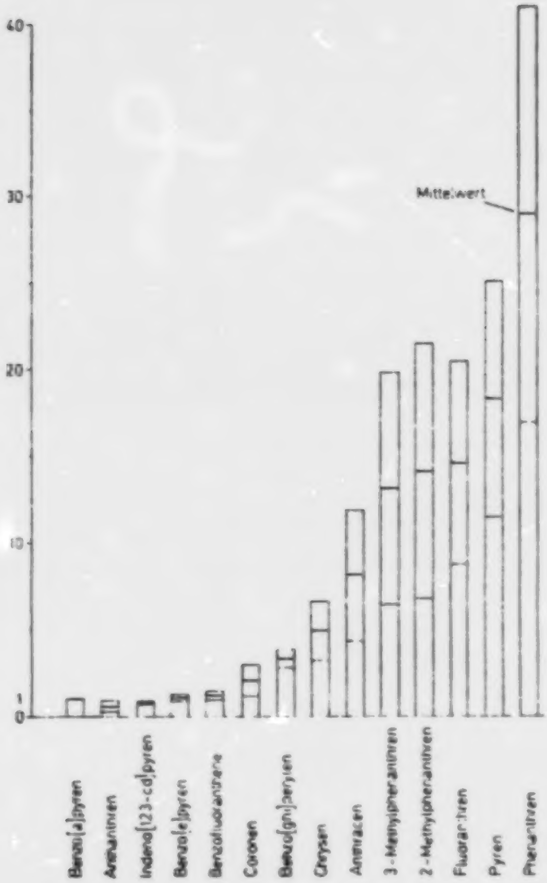


FIGURE 71

Profile of the Emitted PAH (amount of emitted PAH relative to emitted benzo(a)-pyrene). EUROPA-test; 100 cars.

(Source: Reference 114)

Grimmer (116) reported on the PAH content of gasoline automobile exhaust condensates. The particulate matter of the exhaust, collected during a Europa drive cycle, accounted for 90 - 95% of the carcinogenic activity of the exhaust with benzo(a)pyrene accounting for 5 - 10% of the activity. Table 49 summarizes the PAH content of exhaust condensates determined in this study. The PAH content of the exhaust condensate was determined by differential extraction with separation on a Sephadex LH column and qualitative and quantitative analysis using gas chromatography, by which technique more than 100 PAH's were identified in the exhaust samples. Six previously unknown PAH's were identified in the exhaust and, based on the fact that the 6 known carcinogenic PAH's appeared to contribute about 10 -15% of the total carcinogenicity, it was suggested that the unknown compounds were the primary carcinogenic components.

TABLE 49

PAH Content of Exhaust Condensate (mcg/g)

A.	Benzo(a)pyrene		340
B.	Benzo(a)anthracene		280
	Chrysene		410
	Benzo(b)fluoranthene		162
	Benzo(j)fluoranthene		94
	Diben(a,h)anthracene		96
	Indeno(1,2,3-cd)pyrene		268
C.	Cyclopenteno(cd)pyrene	Approx	2000

(Source: Reference 116)

A number of other reports have been published or presented at international meetings in which the fractionation and identification of various components of diesel exhaust particulate have been elaborated. Detailed discussions of these reports would serve little purpose, but references are provided for the convenience of the readers (4, 117).

The voluminous data published thus far on the PAH content of diesel exhaust clearly indicates that, at least qualitatively, the PAH species present in the exhaust from both small and large diesel engines are similar. Because of varying methodology and the multitude of factors which affect PAH emissions, the quantitative data is of relative value and provides orders of magnitude. The identification of PAH's in diesel and spark ignition engine exhaust which are considered to have carcinogenic and/or mutagenic potential has been clearly established.

In contrast to diesel exhaust, it was found that airborne particulates collected from several areas in New York State using high volume samplers contained qualitatively similar amounts of most parent PAH's with 3 - 7 rings. Very few alkyl substitued PAH's were detected. The sample collection methods were different and a strict comparison between airborne PAH's and those in diesel particulate was thought to be inappropriate at the time.

Table 50 gives the concentration of PAH's in the Sudbury air during the winter of 1975-76 and 1980-81. Table 51 provides similar data for Toronto, Hamilton and Sarnia for the 1975/76 period. Of the 7 PAH's measured, four are considered to be carcinogenic and include benzo(a)pyrene, naphtho(1,2,3,4,d,e,f)chrysene, benzo(r,s,t)pentathene and benzo(g,h,i)perilene. The highest values were measured in Hamilton and the lowest in Sarnia (454).

Katz (455) measured the concentration of PAH's in the ambient air of four Canadian cities and found levels as high as 10,000 ng/1000 m^3 of such carcinogens as benzo(g,h,i)perilene, i.e. 0.01 mcg/m^3.

Discussions of additional comparative data of ambient air and the underground mining environment will be given in Section 4.6.

Table 50

Concentration of PAH's in Sudbury air, winter 1975-76 and 1980-81, ng/1000 m^3

Site	Sudbury Oct-Dec 1975(1)	Sudbury Jan-Mar 1976(1)	Sudbury winter 6 hr 1981-82	Sudbury winter 6 hr 1981-82	Sudbury winter 18 hr 1981-82	Sudbury winter 24 hr 1981-82	Sudbury winter 24 hr 1981-82	Average Sudbury winter 1981-82	Mine site surface air	Mine intake air
Benzo(a)pyrene	342	444	150	370	250	260	20	210	20	80
Benzo(e)pyrene	255	317	3760	4950	2740	650	840	2588	840	2800
Benzo(b)fluoranthene	417	650	--	--	--	--	--	--	--	--
Benzo(k)fluoranthene	197	271	380	830	790	600	110	542	400	150
Perylene	41	50	10	110	100	80	10	62	10	20
Dibenz(def,mno)chrysene	37	32	10	110	60	60	10	50	10	50
Benzo(ghi)perylene	2321	3009	440	1060	300	630	70	500	90	1000
Naptho(1234,def)chrysene	99	230	180	3590	590	1050	100	1102	170	1600
Benzo(rst)pentaphene	17	36	100	280	140	90	30	128	20	90
Dibenzo(b,def)chrysene	54	130	--	--	--	--	--	--	--	--

(Source: Reference 454)

Table 51 Concentrations of 7 PAH's in Toronto, Hamilton and Sarnia Air, Winter 1975-76, ng/1000 m^3

Site	Toronto freeway Oct-Dec 1975(1)	Toronto freeway Jan-Mar 1976(1)	Average Toronto freeway winter 1975-76	Toronto suburban Oct-Dec 1975(1)	Toronto suburban Jan-Mar 1976(1)	Average Toronto suburban winter 1975-76	Hamilton urban-industrial Oct-Dec 1975(1)	Hamilton urban-industrial Jan-Mar 1976(1)	Average Hamilton urban-industrial winter 75-76	Sarnia urban-industrial Oct-Dec 1975(1)	Sarnia urban-industrial Jan-Mar 1976(1)	Average Sarnia urban-industrial winter 75-76
*Benzo(a)pyrene	1674	720	1197	729	814	772	3498	1934	2716	596	190	393
Benzo(k)fluoranthene	916	508	712	597	519	558	5145	443	2794	439	104	272
Perylene	193	108	150	136	51	94	403	347	375	87	19	53
Dibenz(def,mno)chrysene	234	34	134	102	162	132	389	150	270	44	7	26
*Benzo(ghi)perylene	10528	4413	7470	4693	9814	7254	7532	6418	6975	2700	1158	1929
*Naptho(1,2,3,4,def)chrysene	638	228	433	300	2762	1531	2027	903	1465	434	129	282
*Benzo(rst)pentaphene	150	67	109	90	259	174	434	281	358	69	23	46
Total			10158			10515			14953			3001
Total carcinogens			9158			9731			11514			2650

*Carcinogens

(Source: Reference 454)

3.4.9 Acid Mists

Concerns for the presence of acid mists in mining atmospheres appears to be relatively recent. The presence of NO_X and SO_2 in a humid atmosphere can lead to the formation of HNO_3 and H_2SO_4, respectively.

Although an estimate of the presence of acid in the mine environment will be made in a later section, a summary of diesel-related acid production is given here.

The formation of SO_2 by diesel engines is directly related to the sulphur content of the fuel, therefore reducing the fuel sulphur content would result in a lowered potential for acid formation. Of particular concern are the recent findings of an ORF (93) study showing that although platinum catalyst converters reduced SO_2 emissions, they resulted in increased H_2SO_4 levels in diesel exhaust. It was estimated that some 35 - 45% of the SO_2 is converted to H_2SO_4 by a catalytic converter. On the other hand, a water scrubber, alone or in tandem with the converter, removed the SO_2 from the exhaust.

This effect of platinum converters has been recognized for gasoline engines for some time (see reference 120). In the latter report, 97% of the sulphuric acid was found to be associated with respirable particulate of less than 0.3 u aerodynamic diameter. As will be discussed in Section 3.9.8 sulphuric acid has been found to be associated with respirable mine dust (121).

The Matheson Gas Data Book (122) and other references provide little information on the rates and ease of reaction of SO_2 with water other than to state that sulphurous acid is formed. Sulphur dioxide has a solubility in water of about 10% (by weight) at 20°C and 1 atmosphere.

According to the Matheson Gas Data Book (122), NO_2 reacts with water forming nitric and nitrous acids, the latter decomposing, especially at higher temperatures, to nitric acid and NO. However, NO_2 undergoes appreciable thermal decomposition at temperatures above 600°C (1112°F) to NO and O_2, which suggests that at higher operating modes, diesel-related HNO_3 formation probably would not be significant.

Nitric oxide (NO), which has a relatively high water solubility (7.3 ml/100 g water @ 0°C, 1 atm), is not converted directly to HNO_3, but is slowly oxidized to NO_2 in the presence of oxygen (see Section 3.9.2 for a more detailed discussion of the latter reaction).

3.5 Exhaust Modifiers

3.5.1 Fuel Additives and Smoke Suppressants

Information from engine manufacturers, mine operators and inspectors and CANMET engineers indicate that fuel additives, such as smoke suppressants, and cetane improvers are not used and thus a detailed discussion of this topic is not included in this report.

The 1977 - NIOSH Workshop concluded that "smoke suppressant fuel additives were neither widely used nor essential to diesel operation". They recommended that smoke suppressant additives not be permitted because of their detrimental effect on engine life and their adverse effect on particulate emissions.

3.5.2 Catalytic Converters and Water Scrubbers

A major engine manufacturer (123) recommends that catalytic afterburners and exhaust gas cleaners (water scrubbers) not be used. Despite this "authoritative" view, the present authors found through mine, Provincial inspection and CANMET personnel that most, if not all, diesels used in Canadian mines are operated with some sort of exhaust modifying equipment. A similar determination was voiced by the 1977 - NIOSH Emissions Workshop which estimated that about 60% of U.S. mine diesels used oxidation catalysts and 40% used water scrubbers.

A major objection to water scrubbers is the need to maintain adequate fluid in them in order to retain their efficiency. From conversations with mining and inspection personnel, there appears to be some doubt that equipment operators pay sufficient attention to this point. It may be necessary to designate a single individual to service engines equipped with water scrubber devices on a regular, recorded schedule to ensure proper operation. An Ontario Research Foundation (ORF) (93) scrubber performance study showed that reasonable efficiency, in terms of particulate and SO_2 capture and total hydrocarbon (THC) reduction, remained even when the scrubber was operated 1/3 full.

Although maintenance problems may be fewer with catalytic converters than with water scrubbers, the former suffer from the disadvantage of being dependent on the exhaust temperature for efficient operation. At the scrubber inlet of LHD and diesel locomotives, the exhaust gases have a temperature of 500° - 800 °F (260 - 427°C) during normal working conditions. During idle, the exhaust temperature rapidly falls to 250° - 300°F (121 - 149°C) (38).

At lower exhaust temperatures, unburned oil and carbon is deposited on the platinum bed thus reducing the effectiveness of the converter. However, during subsequent normal load and speed operation this accumulation is "burnt off", thus returning the converter efficiency to normal.

Nevertheless, reducing idle time of equipment with catalytic converters is beneficial. The most efficient oxidation is when the catalyst inlet temperature is 415°C (213°C) or higher, although oxidation begins at lower temperatures. For example, for some engines a 50% oxidation is attained at 350°F (177°C). Data from the ORF investigation (93) confirmed that although the effects derived from the use of a platinum converter varied with exhaust temperature there were, nevertheless, significant benefits derived at all operating conditions examined, which included cool operation at 200°C - 390°C.

A concern of some importance is the finding that the treatment of exhaust with a platinum catalyst leads to the conversion of a significant portion of the SO_2 to H_2SO_4. This results in a fine "acid" mist which is respirable by itself and by adsorption onto diesel and other particulate matter present in the mine atmosphere.

Although the general literature (see references (8, 124, 125, 126) contains some information on the effectiveness of catalytic converters and water scrubbers, possibly the most recent and relevant data are available from the ORF study referred to above (93).

Using a Deutz V-6 IDI diesel engine, typical of those used on LHD equipment, Lawson and Vergeer (93) evaluated the effectiveness of several exhaust scrubbers using the CANMET load-speed variations described earlier (Section 3.3.5). They evaluated a single pass water scrubber (full and 1/3 full), a packed-bed water scrubber and a catalytic converter. The effect of having the catalytic converter in series with the packed scrubber was also studied.

Table 52 summarizes the ORF findings.

TABLE 52

Average Removal Efficiencies (%) For Various Exhaust Modifiers*

	Full Water Scrubber	1/3 Full Water Scrubber	Packed Scrubber	Catalytic Converter	Converter Plus Packed Scrubber
CO	0	0	0	90	48
NO	0	0	0	25	88
NO_2	0	0	0	-25**	-17
Particulate	31	31	22	0	48
SO_2	53	39	66	72	91
H_2SO_4	-65	-65	0	-425	-265

(*) For a typical LHD cycle for a 213 metre haul, using 0.25% sulphur fuel.

(**) - = increase in concentration in exhaust

(Source: Reference 93)

It is interesting to note that although the catalytic converter by itself removed none of the soot, the use of a converter plus a packed scrubber in series was somewhat more efficient than the scrubber alone. This may have been the result of an increase in the hydrophilic character of the soot as a result of hydrocarbon removal by the catalytic converter. The effect of engine rpm on soot removal efficiency for the different systems studied can be seen in Figure 72. As could be predicted, no CO was removed by the wet scrubbers, whereas the catalytic converter removed, on average, about 90% of the pollutant. This removal efficiency was approximately the same at all engine loads. At all loads and rpm test conditions the raw exhaust contained NO at concentrations of around 600 to 700 ppm and no NO_2. Neither the water scrubber nor the packed scrubber removed any NO, whereas the catalytic converter removed about 25% of the exhaust NO. The combination of a converter and a packed scrubber was somewhat less efficient with an average 17% removal of NO. The catalytic converter did, however, result in the oxidation of a fraction of the NO to NO_2 so that there was an increase in the NO_2 content of the exhaust of 20 -25% in the presence of the catalytic converter. The presence of the water scrubber downstream from the catalytic converter, although not removing any of the NO_2, did appear to reduce the amount of conversion occurring at the catalytic converter. The removal of nitric oxide by the catalytic converter varied with the engine load as shown in Figure 73. The use of a wet scrubber, either as a single pass or packed unit, removed 40 - 70% of the exhaust SO_2. The larger surface area in the packed scrubber appeared to make it more efficient at removing SO_2, particularly at full load. The monolithic catalytic converter also appeared to remove significant quantities of SO_2, but this was partially as a result of the conversion of SO_2 to sulphate (Figure 74). A wet scrubber, placed downstream from the converter, removed about half of the formed sulphate plus virtually all of the residual SO_2 (Figure 75).

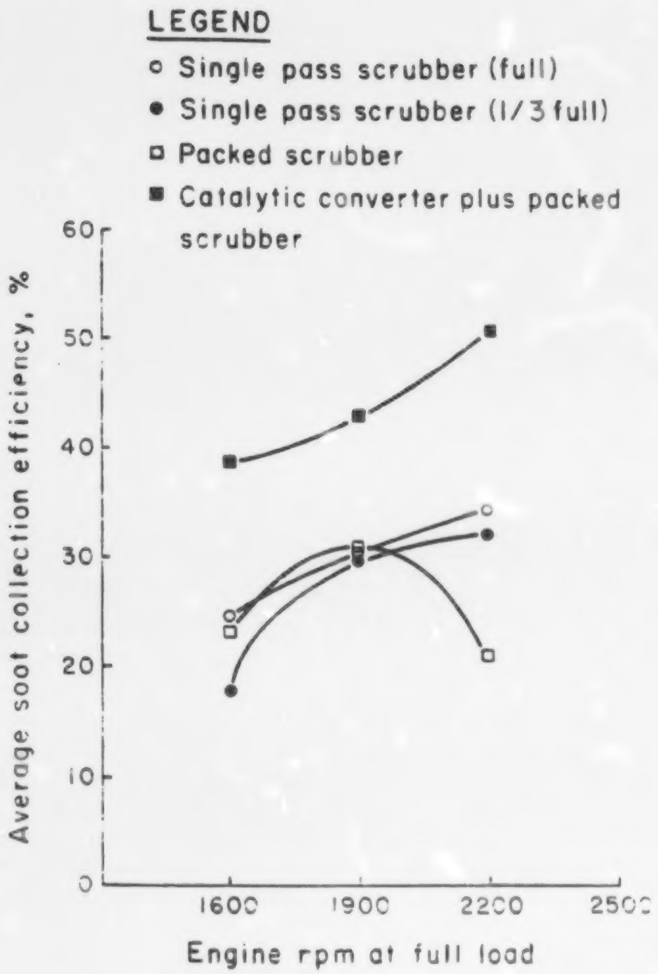


FIGURE 72

Scrubber soot performance

(Source: Reference 93)

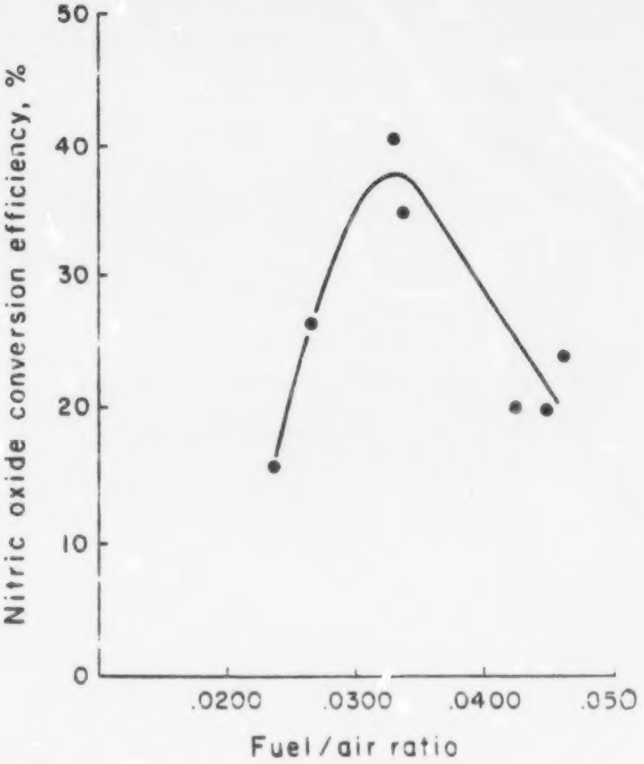


FIGURE 73

Catalytic purifier nitric oxide performance

(Source: Reference 93)

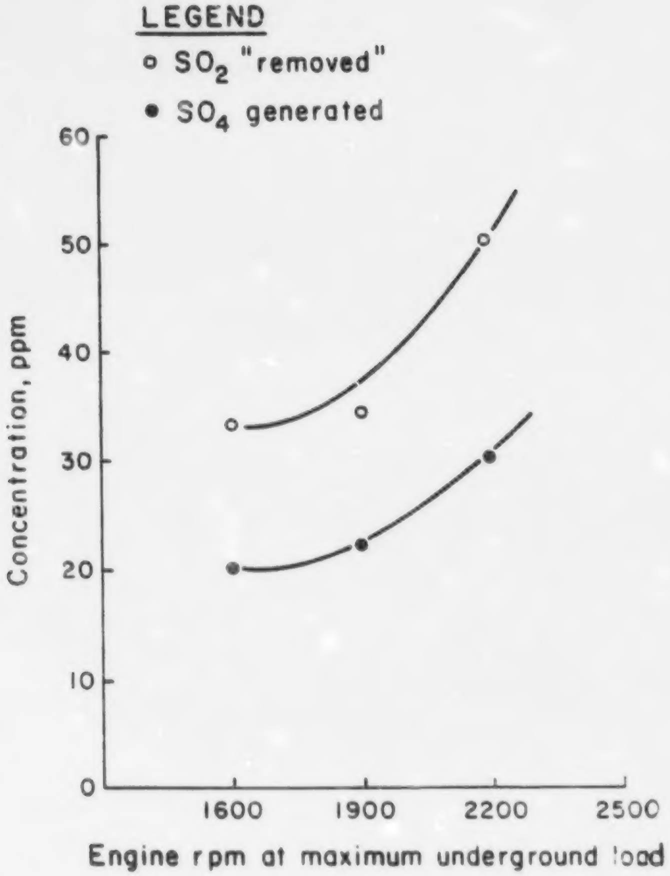


FIGURE 74

Catalytic converter sulphur oxides performance

(Source: Reference 93)

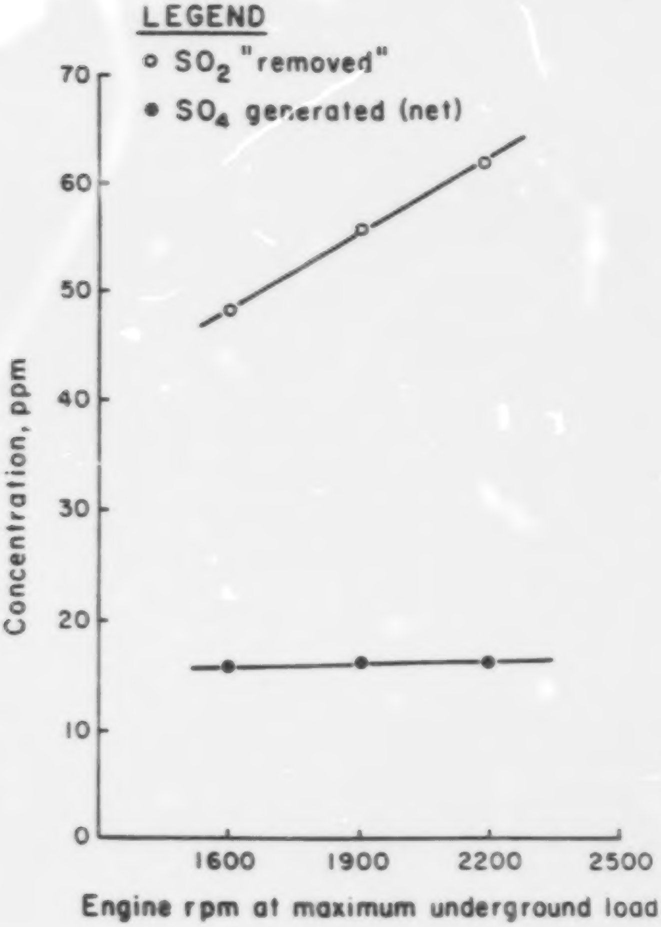


FIGURE 75

Catalytic converter plus packed scrubber sulphur oxides performance

(Source: Reference 93)

Water scrubbers, by acting as heat exchangers, are used on diesels in U.S. coal mines to lower the exhaust temperature to prevent mine fires.

At the 1977 - NIOSH meeting, the raw and treated exhaust values in Table 53 were suggested as being typical for LHD vehicles used underground. These values may have been predicated mainly on the ORF data supplemented by participant knowledge and manufacturers' information. Nevertheless, it contains the considered opinion of the experienced members of the Emissions Work Group and thus may be considered as a summation of the best available information at the time.

Perusal of the ORF data would suggest that maximum benefits would be derived from the use of a combination of a catalytic converter followed by a wet scrubber. This should lead to decreased particulate output and lower emission levels of CO, SO_2, THC and NO_2. In addition, the presence of the water system could reduce the H_2SO_4 output caused by the catalytic converter.

Hunter and co-workers (127) evaluated a medium duty Caterpillar 3208 direct injection diesel engine equipped with an Engelhard PTX Series diesel oxidation catalyst during 6 modes of the EPA 13-mode heavy-duty cycle on the output of total particulate, soluble organic fraction, sulphates, NO, NO_2, NO_X and hydrocarbons. The mutagenicity of the SOF sub-fractions using the Ames assay were also examined. It was found that brake-specific NO_X emissions remained unchanged with the use of the catalyst, but the NO_2 emissions were increased at most modes with a similar decrease in NO emissions. Brake-specific hydrocarbon and SOF emissions were both decreased by the use of the catalysts. The percent reduction of these species was relatively unaffected by changes in engine speed or load. Sulphate emissions were greatly increased by the catalyst at all test modes. The SO_4 increase was due to the oxidation of SO_2 and SO_3 with their eventual release as sulphates. Particulate emissions were generally increased by the catalysts as a result of an increase in sulphate emissions and an increase in the solid (unextractable) portion of the particulate.

TABLE 53

Estimates of Diesel Engine Exhaust Emission Factors and Exposures[1]

	Exhaust Emission Estimates (g/bhp-hr)			
Component	Untreated Engine	Catalyst Treated Engine	Scrubber Treated Engine	Catalyst and Water Scrubber Treated Engine
CO	0.6 - 2.7	0.6 - 0.3	0.6 - 2.7	0.6 - 0.3
CO_2	510 - 600	510 - 600	510 - 600	510 - 600
Hydrocarbons	0.03 - 0.17	0.03 - 0.017	0.02 - 0.12	0.003 - 0.017
Benzene[a]	1.5×10^{-4} to 1.7×10^{-3}	1.5×10^{-5} to 1.7×10^{-4}	1×10^{-4} to 1.2×10^{-3}	1.5×10^{-5} to 1.7×10^{-4}
NO	1.25 - 4.1	1.25 - 3.5	1.25 - 4.1	1.25 - 3.5
NO_2	0.3 - 0.7	0.15 - 1.1	0.3 - 0.7	0.15 - 1.1
SO_2	0.5	0.25	0.096	0.24
Particulate[4]	0.17 - 0.67	0.17 - 0.67	0.12 - 0.47	0.08 - 0.33
Aldehydes[5]	Footnote[6] 0.02 - 0.20[b]	Footnote[6] 0.005[b]	Footnote[6] 0.01[b]	Footnote[6] 0.005[b]
Formaldehyde	Footnote[6]	Footnote[6]	Footnote[6]	Footnote[6]
Total Aldehydes	0.02 - 0.2	0.005	0.01	0.005
H_2SO_4	----	0.37	----	0.24
Aromatics	Footnote[6]	Footnote[6]	Footnote[6]	Footnote[6]
PNA[2]	----	----	----	----
Phenols	0 - 0.01	2×10^{-3} to 1×10^{-3}	?	2×10^{-3} to 1×10^{-3}

---

[1] Table prepared from selected information presented at NIOSH Workshop on Diesels in Underground Coal Mines, Sept. 19-24, 1977, Morgantown, W. Va.

[2] NIOSH workshop consensus concluded that no reliable estimates existed.

[3] Exhaust emission estimates are for SO_X.

[4] Carbon only (NIOSH workshop estimated that carbon represented approximately 80% of total diesel particulate).

[5] Excluding oxygenates (this footnote was not in original text of the workshop).

[6] No exhaust emission estimates made at the workshop.
a) Changed to read 0.5 - 1.0% of hydrocarbons in final write-up of workshop.
b) Values added in final write-up of workshop.

(Source: Reference 10)

The use of the catalyst generally resulted in a greater mutagenic specific activity of the SOF. At some modes, the increase in the biological activity of the SOF was sufficient to offset the reduction in mass emissions. Table 54 provides a list of the compounds tentatively identified in the Mode 11 aromatic subfraction. Compounds 4 - 8 and 13 are known mutagens.

McMahon et al (128) reported on the use of an exhaust system on passenger diesel vehicles which employed 2 alumina-coated, stainless-steel-wool-coated cartridges inserted into an outer housing about the same size as a conventional muffler. This system removed about 70% of the particulate and significant quantities of hydrocarbons and sulphates. A 50% reduction in polynuclear aromatics, measured as BaP, was also effected. Because of the high soot generated by diesel engines regeneration at temperatures above 1000°F is necessary every 150 to 200 miles. Additional development work to conveniently affect this regeneration procedure is on-going.

The concept of using a catalytic ignition and combustion system for the disposal of diesel soot has been presented by Hilldenbrand and Trayser (118) and Murphy et al (129). Using a trap-oxidizer concept, the soot was collected and catalytically treated to make ignition and combustion more practical under diesel engine exhaust conditions. The design of these units is aimed mainly at light-duty diesel engines. Solutions of metal salts such as copper, cobalt, etc., are effective for lowering ignition temperature. Mixtures of copper chloride and sodium chloride were especially effective and applications of solutions containing these salts have been used to achieve ignition. The results were encouraging for a method in which highly controlled burn-off of trapped soot was achieved with minimal extra energy requirements. The practical application still requires development of suitable hardware and identification of the optimum operation cycle for the trap/oxidizer (118, 129, 130). These approaches were critically reviewed by Williams (89).

TABLE 54

Tentative Identities of Compounds in Mode 11 Aromatic Subfraction as Determined by Gas Chromatography-Mass Spectrometry

Peak Number (figure)	Retention Time (min.)	Compound	Molecular Weight
1	19.2	2,6-di(t-butyl)-4-methyl phenol	220
2	28.6	Phenanthrene	178
3	31.2	Monomethyl dibenzothiophene isomer	198
4[a]	32.0	Monomethyl dibenzothiophene isomer	198
5[a]	32.8	Monomethyl phenanthrene isomer	192
6[a]	33.0	Monomethyl phenanthrene isomer	192
7[a]	33.6	Monomethyl phenanthrene isomer	192
8[a]	33.8	Monomethyl phenanthrene isomer	192
9	34.8	Dimethyl naphthothiophene isomer	212
10	35.6	Dimethyl naphthothiophene isomer	212
11	36.3	Dimethyl naphthothiophene isomer	212
12	37.6	Dimethyl phenanthrene isomer	206
13[a,b]	38.3	Fluoranthene	202
14[b]	39.9	Pyrene	202
15	41.2	Trimethyl phenanthrene isomer	220
16	48.0	Benzo(ghi)fluoranthene	226
17	53.0	Phthalate ester (insignificant mass ion)	--

[a] Known mutagens (7)

[b] Despite nearly identical mass spectra, identity of these compounds was confirmed by retention times

(Source: Reference 127)

Mogan et al (454) reported on a collaborative study with Westaway of Laurentian University, Sudbury, Ontario, in which the effects on PAH output of a monolithic catalyst (PTX -Compact Honeycomb) or a pelleted catalyst supported on aluminum spheres were compared using a direct-injection Caterpillar engine. Table 55 gives detailed data of the concentration of 12 PAH's in the atmosphere of a hard-rock mine in the vicinity of a diesel-powered LHD with no exhaust treatment equipment, and when equipped with a monolithic catalytic purifier or a pelleted purifier. It will be noted that on average, both the 7 non-carcinogenic and 4 carcinogenic PAH's increased only slightly with the use of the monolithic converter and all were substantially reduced by the use of a pelleted catalytic converter. Although this data was encouraging, particularly as it related to the pelleted converter, when the diesel soot was extracted with dichloromethane and subjected to an Ames mutagenic assay, the results were much less encouraging. As can be seen in Table 56, compared to untreated exhaust samples, the concentration of mutagens in the atmosphere of the hard-rock mine during the use of a monolithic catalytic converter increased some five-fold and with the pelleted catalyst, over 30-fold. It was postulated that the lack of correlation between PAH concentrations and mutagenic activity was related to the presence of dinitropyrenes, which are among the most potent mutagens identified to date. Because they were not analyzed in the exhaust particulate samples they would show up in mutagenicity test but not in the quantitative analysis. Additional discussion of the PAH content and mutagenic activity of the atmosphere in a hard-rock mine may be found in Sections 4.6 and 3.9.4.

3.6 Uranium Mining and Radiation Exposure

3.6.1 Introduction

It has been known for many years that excessive exposure to radiation during uranium mining can result in an increased incidence of lung cancer in miners (131). As was detailed in the opening chapter of this report, some Canadian uranium mines employ diesel equipment underground. The uranium miner in this situation may, in a sense, be

Table 55: Concentrations of Twelve PAH's in the Atmosphere of a Hardrock Mine in the Vicinity of a Diesel Powered LHD with no Exhaust Treatment, a Monolithic Catalytic Purifier, and a Pelletted Purifier

	1 No exhaust treatment 1980-81	2 No exhaust treatment 1980-81	3 No exhaust treatment 1980-81	ii Monolithic catalyst 1980-81	iii Monolithic catalyst 1980-81	6,7 Monolithic catalyst 1980-81	8 Pelletted catalyst 1980-81	9 Pelletted catalyst 1980-81	10 Pelletted catalyst 1981-82	11 Pelletted catalyst 1981-82	12 Pelletted catalyst 1981-82	13 Pelletted catalyst 1981-82
Benzo(a)pyrene	862	546	1566	1197	792	387	96	56	280	650	290	340
Benzo(k)fluoranthene	992	870	1720	802	771	739	262	188	460	1250	860	1210
Perylene	108	72	218	216	160	103	48	24	110	190	130	210
Dibenzo(def,mno)chrysene	96	90	145	202	179	155	10	14	200	200	220	200
Benzo(ghi)perylene	3248	1118	2470	4126	3898	3670	1370	182	1480	1040	1480	1920
Naptho(1234,def)chrysene	5037	1442	774	634	3798	6965	494	254	2350	2260	1910	2580
Benzo(rst)pentaphene	553	524	48	165	168	171	112	89	290	620	530	440
Pyrene	42981	12380	10645	18658	15327	11996	16327	20585	57900	30600	76500	109000
Fluoranthene	1289	2761	492	1209	1768	2326	3160	454	4520	8730	3800	11400
Benzo(a)anthracene	4505	1683	900	1034	1570	2106	722	1039	28700	14800	25300	10900
Benzo(e)pyrene	25025	1226	7388	2530	3181	3831	8175	3178	9770	5460	280	16200
ΣPAH's (7)	10487	4993	6982	7342	9766	12190	2392	807	5170	6210	5420	6900
Σcarcinogens (8)	9731	2961	4879	6122	8656	11193	2072	561	4400	4570	4210	5280

(Source: Reference 454)

Table 56: Concentration of Mutagens in the Atmosphere of a Hardrock Mine in the Vicinity of a Diesel Powered LHD with no Exhaust Treatment a Monolithic Catalytic Purifier, and a Pelletted Purifier, and in a Salt Mine in the vicinity of a Loader with a Monolithic Purifier (14 and 14)

	2 No exhaust treatment 1980-81	4 No exhaust treatment 1980-81	6 Monolithic catalyst 1980-81	7 Monolithic catalyst 1980-81	8 Pelletted catalyst 1980-81	9 Pelletted catalyst 1980-81	10 Pelletted catalyst 1980-81	11 Pelletted catalyst 1981-82	12 Pelletted catalyst 1981-82	13 Pelletted catalyst 1981-82	14 Monolithic catalyst 1981-82	15 Monolithic catalyst 1981-82
Specific activity TA98 minus S9 revertants/µg	0.25*	0.18*	2.6	3.8	66	210	57	39	79	75	2.8	0.95
Extractables per filter, mg	17.1	50.2	69	54.3	37.2	14.9	20.9	11.4	28.4	54.9	6.6	8.0
Mine air, volume through filter, m^3	224	108	465	411	444	400	129	116	117	158	19	13
Concentration of mutagens in mine air revertants/m^3	19	84	390	500	5500	7800	9200	3800	19000	26000	970	580

*Values of 6.2 and 11 were also measured; the values quoted in the Table are supported by unpublished dynamometer studies at the Ontario Research Foundation.

(Source: Reference 454)

in double jeopardy. That is, he is exposed to the potential adverse effects of both diesel exhaust and ionizing radiation. As will be discussed in the health section, if a miner is a smoker, he is in triple jeopardy or greater; greater because it is a possibility, although not quantifiable at this time, that some of the exhaust constituents, radiation and smoking, have synergistic toxic effects (132, 133).

Thus, the inclusion of a review of the radiation chemistry related to uranium mining is justified. Of the many diverse areas and topics requiring review in the preparation of this document, the authors found the complexities of the physical, chemical and biological properties of uranium radiation one of the more difficult to assemble, comprehend and report.

In order to assist others who may find the topic equally difficult to understand, the following review is included as background to the discussion of the health effects of radiation.

The authors have attempted to keep the mathematical aspects of the topic to a minimum, and readers are directed to references, such as 131, 134 and 135, for additional details.

3.6.2 Definitions

A prerequisite to discussing the broad topic of radiation, radon, radon daughters, etc., is the need to understand several definitions, which follow:

Ionization:

The most important effect that radioactive substances have is to cause ionization of matter. Ionization occurs when an atom loses an orbital electron, which leaves the atom positively charged. The presence of the positive ion and the negative electron together is referred to as an ion pair.

There are three basic types of ionizing radiation, which can be briefly described as follows:

Gamma (γ) Rays

Gamma radiation is an electromagnetic ray coming from an unstable atomic nuclei. They have definite energies which are characteristic of the substance they come from. Of the three types of ionizing radiation, gamma rays have the greatest penetrating power.

Beta (β) Particles

Beta particles are fast-moving electrons which are produced when a neutron is spontaneously changed to a proton in the nucleus of an atom containing too many neutrons. They have spectrums of energy rather than discrete energies, and are less penetrating than gamma rays, but are more penetrating than alpha particles. They can be absorbed in a few mm of aluminum.

Alpha (α) Particles

The alpha particle has the same structure as a helium nucleus, with two neutrons and two protons. They mainly result from the radioactive decay of heavy elements such as uranium and radium. Each alpha particle has its own discrete energy. They have great ionizing power and can be very destructive, but their penetration is short compared to beta and gamma radiation. For example, a sheet of paper will stop an alpha particle.

Alpha and beta particles have such weak penetrating power that they are only harmful when deposited internally, whereas gamma rays have deep penetrating characteristics and can be harmful from external as well as internal sources.

Certain definitions, given below, may be of assistance in understanding the complexities of uranium radiation:

Half-Life: Radioactive materials decay or emit less radioactivity with time. The time required for the number of radioactive nuclei to decrease by a factor of two is referred to as the half-life ($t_{\frac{1}{2}}$) of the radioactive substance, and is an exponential event. The $t_{\frac{1}{2}}$ can vary from 10^{-6} sec or lower to billions of years.

Curie (Ci): A unit used to express radioactivity. It was originally related to the activity of radium, but is presently defined as 3.7×10^{10} nuclear disintegrations per second (dps). A millicurie (mCi) and microcurie (uCi) are 10^{-3} and 10^{-6} curies, or 3.7×10^{7} and 3.7×10^{4} dps, respectively.

Specific Activity: Refers to the radioactivity (Ci) per gram of radioactive material.

Roentgen (R): Unit of exposure dose related to the amount of ionization produced under specific conditions. It is the quantity of radiation which creates 2.08×10^9 ion pairs per cc of air at STP. (The R is not a unit of ionization and applies only to alpha and gamma radiation).

Rad (radiation absorbed dose): A measure of the energy given to matter by ionizing radiation per unit mass in any medium. A unit of absorbed dose. One rad is equal to the deposition of 100 ergs of energy per gram. The rad unit is applicable to all types of radiation.

RBE (relative biological effectiveness): A term used to define the difference in biological damage caused by different ionizing radiations when administered at the same rad dose.

Large mass particles, with relatively slow speed (i.e. alpha particles), give up more energy along their path of travel than beta particles and therefore alpha particles cause more damage. Usually, beta particles (electrons) and gamma rays are given an RBE of 1. An RBE of 10 is used for alpha particles.

Rem (roentgen-equivalent-man): A dose equivalent which is related to the rad by the equation:

$$\text{rem} = \text{rad} \times \text{RBE}$$

It is the unit used to express the dose of radiation received by workers. Obviously, for gamma rays with an RBE of 1, the rem dose and rad dose are the same.

Electron Volt (eV): The unit used to describe the energy of radiation. It is a unit of energy equal to that gained by an electron when it is accelerated through a potential of 1 volt.

3.6.3 Uranium Radiactivity

Detailed reviews of uranium radiation and radon daughter behaviour can be found in standard texts and in references 131 and 134.

Ore or rock that contains uranium emits all three types of ionizing radiation - alpha, beta and gamma.

Natural uranium contains:

Uranium-238 (^{238}U) - 99.3%
Uranium-235 (^{235}U) - 0.7%

Thus, the decay of ^{238}U is of major concern and is shown in Table 57, together with the various types of radiation given off by members of the decay chain, as well as their half-lives.

Natural thorium (^{232}Th) is also present in varying amounts in uranium-containing ore and is the parent of another chain of decay products.

The primary contributor to radioactive exposure in uranium mines results from the alpha particles emitted by radon and its daughter products, which are present in the mining atmosphere.

As is indicated in Table 57, radon-222 is a product of uranium-238 decay. Figure 76 shows a simplified sequence for the decay of uranium-238 to radium-226.

TABLE 57

Radioactive Decay Sequence from Uranium-238 to Lead-206

Historical Name	Isotope	Half Life	Principal Radiations
Uranium I	Uranium-238	4.49×10^9 years	alpha
Uranium X_1	Thorium-234	24.1 days	beta
Uranium X_2	Protactinium-234	1.17 minutes	beta
Uranium II	Uranium-234	2.48×10^5 years	alpha
Ionium	Thorium-230	8.0×10^4 years	alpha
Radium	Radium-226	1602 years	alpha
Radon	Radon-222	3.825 days	alpha
Radium A	Polonium-218	3.05 minutes	alpha
Radium B	Lead-214	26.8 minutes	beta, gamma
Radium C	Bismuth-214	19.7 minutes	beta, gamma
Radium C'	Polonium-214	164 usec	alpha
Radium D	Lead-210	22 years	beta,gamma
Radium E	Bismuth-210	5.02 days	beta
Radium F	Polonium-210	138.3 days	alpha
Radium G	Lead-206	Stable	Stable

(Source: Reference 136)

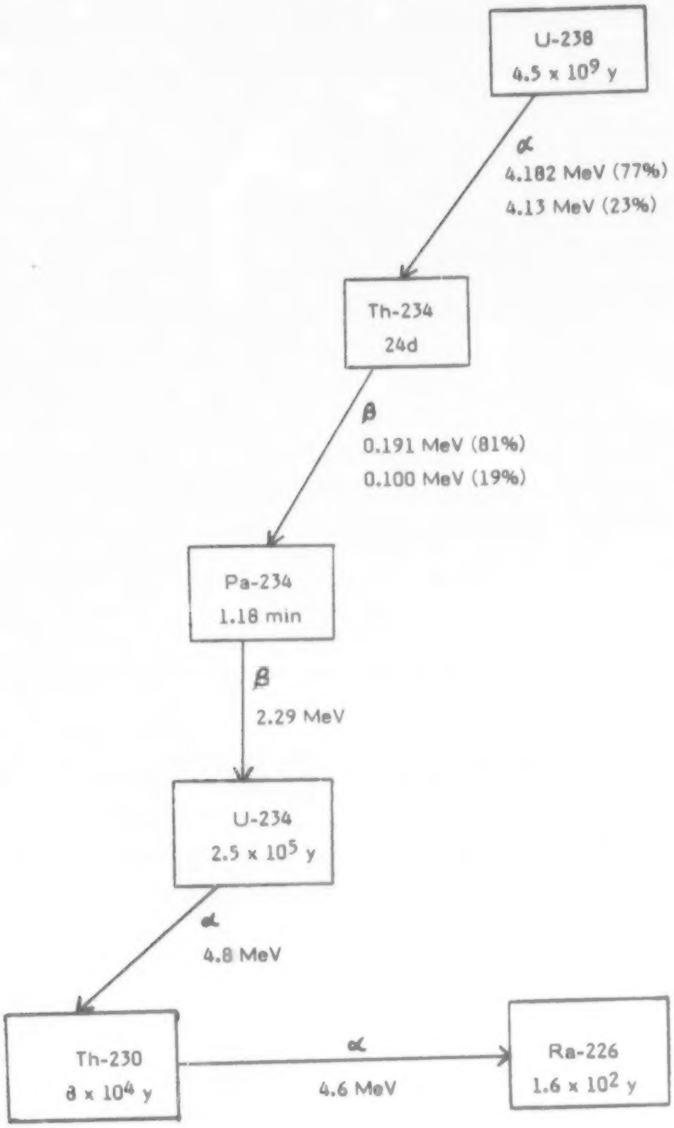

U-238
4.5 x 10⁹ y
α
4.182 MeV (77%)
4.13 MeV (23%)
Th-234
24d
β
0.191 MeV (81%)
0.100 MeV (19%)
Pa-234
1.18 min
β
2.29 MeV
U-234
2.5 x 10⁵ y
α
4.8 MeV
Th-230
8 x 10⁴ y
α
4.6 MeV
Ra-226
1.6 x 10² y

FIGURE 76

Uranium decay sequence
(Source: Reference 137)

The member of the uranium decay chain which is the major contributor to the radiation of miners is, as indicated above, radon-222, which is formed from the last isotope shown in Figure 76, radium-226. — The decay scheme for radon-222, therefore, is of key importance, and is given in Figure 77.

Note in Figure 77 that from polonium-218 to polonium-214, the isotopes RaA, RaB, RaC and RaC' have also been designated. These latter abbreviations refer to what are commonly called radon daughters and they are historical abbreviations for the radon daughters which were once referred to as radium A to radium C'. These latter terms are still in popular use today, although they do not indicate the true element of radioactive isotopes.

Radon-222 is a gas 7.7 times as dense as air which is formed in the ore body or rock and in broken ore body. Radon-222 is very mobile, with a relatively long half-life of 3.8 days and it can migrate through openings or fissures in the rock into the mine atmosphere. Radon has a solubility in water of 230 cm^3/l, and can be absorbed into water present in the mine through seepings or as a result of dust control sprays, etc. With movement of water, the radon can also leave the water and enter the mine atmosphere.

The decay products resulting from the radioactive disintegration of radon-222, referred to above as radon daughters, and including in particular RaA, RaB, RaC and RaC', can be retained in the lungs. In addition, the radon daughters are ionized (carry an electrical charge) when they are formed, and are strongly attracted to airborne dust particles which act as condensation nuclei for water vapour, which can be deposited in the pulmonary system (131, 136, 138).

The two short-lived alpha-emitting daughters, RaA and RaC', are the principal direct sources of radiation dosage to the lung. RaB and RaA decay to RaC' and are therefore indirect sources of radiation.

It has been estimated that the radiation dose to the respiratory system due to alpha decay of inhaled radon daughters is 20 times greater than that due to the decay of radon itself in the lungs (135).

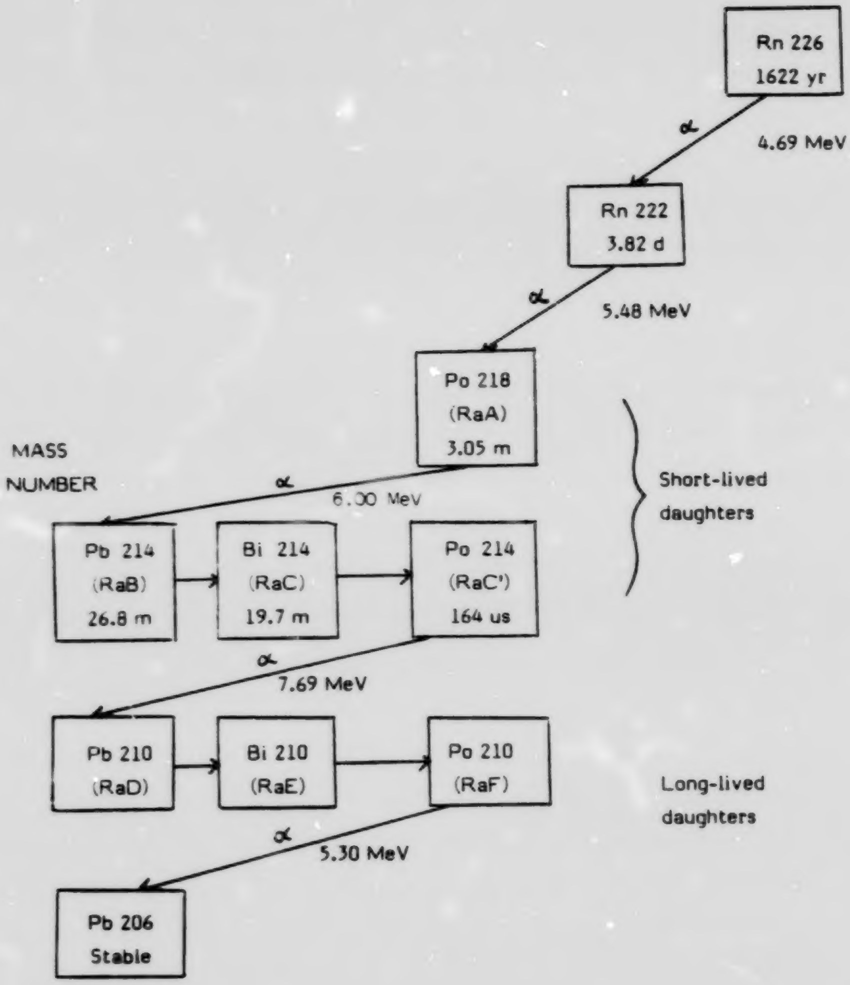

Rn 226
1622 yr
α
4.69 MeV
Rn 222
3.82 d
α
5.48 MeV
Po 218
(RaA)
3.05 m
MASS
NUMBER
α
6.00 MeV
Short-lived
daughters
Pb 214
(RaB)
26.8 m
Bi 214
(RaC)
19.7 m
Po 214
(RaC')
164 us
α
7.69 MeV
Pb 210
(RaD)
Bi 210
(RaE)
Po 210
(RaF)
Long-lived
daughters
α
5.30 MeV
Pb 206
Stable

FIGURE 77

Radon decay sequence

(Source: Reference 136)

The dose of radiation delivered to the lungs is dependent on the concentration of the radon daughters present and the particle size distribution of the dust to which the daughter products are attached (131, 135, 139). Radon daughters that attach or "plate out" on aerosols will follow the behaviour of the particles. Because most particulate in mines is sub-micron in size, essentially all the radon daughters are respirable.

Although discussed in more detail in the particulate (3.4.7) and dust (3.9.6) sections, diesel soot may also act as a carrier of radon daughters which, being virtually 100% respirable, could lead to deposition deep in the pulmonary system.

The free or unattached radon daughters are thought, by some authorities, to be deposited preferentially in the upper intra-thoracic respiratory tract or tracheobronchial region, where most miners' cancer develops (139, 140, 141, 142). The attached atoms, being associated mainly with dust of respirable size, tends to be deposited deeper in the lung.

From a health hazard point of view, more importance is given by many experts to the free or uncombined radon daughters than to radon daughters attached to particulate (131, 135, 139, 141, 143). The present authors feel this point requires further study to determine the relative importance of free and attached radon daughters.

Various investigators (139, 141) have indicated that as the dust concentration increases, the proportion of free radon daughters decreases, and thus the health risk may decrease. As ventilation rates increase, the concentration of unattached atoms increases, thus leading to the possibility of an increased radiation exposure. This leads to the conclusion that the radon health risk in a uranium mine may be greater in areas where no operations are taking place than in working stopes where dust, diesel soot and oil mists are present which could significantly reduce the number of free radon daughters present in the atmosphere.

There is evidence from animal and human retention studies that the elimination rate of deposited Rn-daughters from the lung to the blood is significantly higher than the elimination rate of the dust particles to which the radioactive atoms are attached. This suggests that the adsorbed radioactive atoms must be able to leave their carrier particles by desorption or solution soon after their deposition (139). —

According to Jacobi (144) the ICRP-lung model for soluble aerosols is a reasonable approach to estimating the retention of Rn-daughters in different lung regions. Jacobi pointed out that the biological half-life of 0.5 days of Rn-daughters in the pulmonary region is long compared to the radioactive half-time of the short-lived Rn-daughters. Therefore, it could be expected that nearly 100% of the Rn-daughter activity deposited in the pulmonary region will decay in that region. In the tracheobronchial region, the elimination rate is higher and thus the fraction of the deposited potential energy which is absorbed in this region has been estimated to be 80% for RaA, 40% for RaB and 50% for RaC (144).

The ICRP-lung model permits the calculation of radiation doses to different regions of the pulmonary system. It is, as indicated, dependent on the fraction of daughter atoms inhaled in the form of free (referred to as the f value), unattached atoms or ions.

Figure 78 presents Jacobi's (144) calculations in respect to the ratio between the absorbed alpha-dose in rads and the inhalation exposure in WLM-units as a function of the "v" value for the tracheobronchial (TB) and pulmonary (P) regions of the lungs. It can be seen (Figure 78) that for a low free-atom fraction, fp, the mean bronchial dose is estimated to be higher by a factor of 2 - 3 times than the mean pulmonary or lung dose.

Additional discussions of dosimetry models can be found in various publications from the Lovelace Inhalation Toxicology Research Institute (456).

The present authors are not able to provide a critical discussion of the relative importance of attached and unattached Rn-daughters. However, they feel that more research emphasis should be given to the potential health risk of Rn-daughters which are deposited in the respiratory system attached to respirable particulate. This concern appeared to be voiced by C. N. Davies in a comment to an

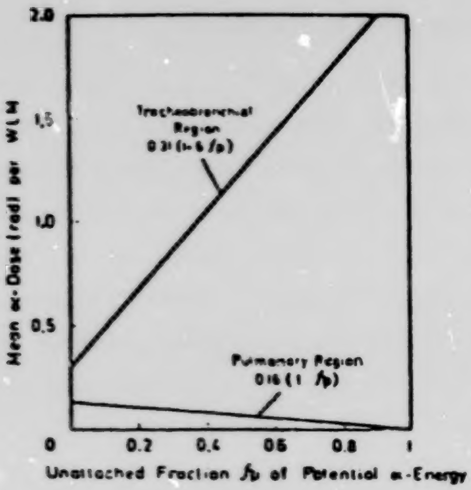


FIGURE 78

Mean alpha-dose per WLM in the tracheobronchial and pulmonary region by inhalation of ^{222}Rn-daughters as function of the unattached fraction of the potential alpha-energy; calculated from the new ICRP-lung model, class D.

(Source: Reference 139)

experimental paper on the topic of "f" measurements by Fusamara et al (140). Davies stated that:

> "You demonstrate that a reduction in the f-value is associated with mining activity, and state that this is due to the production of 'condensation nuclei'. I should prefer to call these 'dust particles'. Such particles, produced in great abundance during operations, collect radon daughter products and pass through the diffusion tube without deposition. They are deposited in the lungs of miners who breathe them in, and constitute a threat to health. I wonder, therefore, whether the measurement of f-values with a diffusion tube is really a valid method of assessing the health risk due to radon daughter products."

Also, the potential for diesel soot to combine with Rn-daughters was emphasized by George and Hinchcliffe (145, 146) in their report of measurements of uncombined radon daughters in uranium mines. They noted that "diesel-powered equipment is one of the principal sources of aerosols in mine air tending to maintain uncombined fractions at low values."

Radon is itself an inert gas, and has a long half-life relative to the time a breath of air remains in the lung. Thus, radon is thought to have a short residence time in the lungs and is exhaled before exposing the lung tissue to significant doses of ionizing radiation. If, however, radon was attached to particulate such as diesel soot (see Section 3.9.6), then radiation exposure from this source might be significant. It could be expected that, as Rn would be carried on respirable sized particulate, that the greatest deposition would occur in the alveolar regions of the lung. The potential for diesel soot to bind radon gas is commented on in Section 3.9.7.

As can be seen in Table 57, Radium B, C and D also give off gamma radiation. This source, together with others, results in gamma exposure to mine workers. The IAEA (International Atomic Energy Agency (135) point out that the level of gamma exposure will vary with the grade of ore and that the frequency of monitoring should be based on the mean exposure. For example, if exposure exceeds 1.5 R per year, it is suggested that quarterly readings be taken rather than annual in estimating worker exposure. If the ore assay is very high, miners should be provided with dosimeters which are read at least monthly.

Although RaB and RaC also give off beta particles, they are considered to be a negligible source of radiation.

The external exposure of the body to alpha particles from radon and its daughters is not considered harmful. The penetrating power of the alpha particle is so low as to be unable to pass the outer layer of dead skin, thus the underlying live tissue is not exposed to the radiation.

3.6.4 Health Effects of Uranium

The health effects of uranium, or more properly, of radon and its daughters, are reviewed in Section 4.12. A few general explanatory comments are included here as background to the sections on maximum permissible limits and methods of radon measurements which follow.

Soluble uranium components are rapidly absorbed into the circulation from the lungs following inhalation. A major portion of the absorbed dose is excreted in the urine with 50 - 75% of the dose being eliminated within 24 hours. A significant fraction of the unexcreted uranium remains in the kidney, and can lead to kidney damage.

Urine analysis for uranium is used to determine the extent of recent internal contamination with uranium sources.

Because of its rapid elimination and propensity to cause kidney damage, exposure limits for soluble uranium have been set more by chemical toxicity considerations than by radiation exposure concerns.

For insoluble uranium compounds the potential for radiation damage, and not chemical toxicity is the factor upon which the MPC has been determined. Inhalation of uranium oxides is a major source of inhaled uranium. The biological half-life is about 120 days and the elimination is not defined by a simple exponential equation. A portion of the uranium is "bound" in bone and is retained more tenaciously than uranium at other sites. Bone is the critical organ for radiation protection control purposes.

Radium is chemically similar to calcium and when ingested deposits in tissues in a manner similar to calcium and is excreted slowly, with a long biological half-life.

3.6.5 Radiation Exposure Limits

The limit of radiation exposures published by the International Commission of Radiological Protection (ICRP) and adopted by the IAEA are as follows:

Organ or Tissue	Yearly maximum (rem)	Quarterly maximum (rem)	Weekly average (mrem)	Hourly average for 40-h week (mrem)
Whole body, gonads, red bone marrow	5	3	100	2.5
Skin of the whole body, bone and thyroid	30	15	600	15
Hands, forearms, feet and ankles	75	38	1500	38
Other single organs	15	8	300	8

(*) Other single organs (bronchial mucosa of lung is taken to be a single organ)

(Source: Reference 135)

It must be emphasized that the ICRP permissible dose represents the dose which, according to the ICRP, has a negligible probability to cause severe somatic or genetic injuries. In other words, if the maximum doses recommended did cause damage it would be of an "acceptable nature or extent".

The table gives both total dose and dose-rate figures. The health assessment is based primarily on the total dose received during a lifetime, whereas the dose-rate is used mainly for control purposes.

For information and comparison purposes only the maximum permissible concentration (MPC) of the uranium isotopes in water, as established by International bodies, are shown in Table 58.

The ICRP have recommended that the MPC of soluble and insoluble uranium dust in air be 6×10^{-11} and 7×10^{-11} uCi/cm^3, respectively. These values have been set based on the toxicity of uranium and not radiation dosage (135).

As was noted earlier, the significant dose of alpha-radiation delivered to the trachea and large bronchi is thought to be a result of radon daughters unattached to dust or condensation nuclei. Using this rationale the ICRP established a formula for the MPC of radon in air

TABLE 58

Uranium Decay Chain - Maximum Permissible Concentration in Water (MPC)

Radionuclide	MPC (uCi/l)	Critical Organ
Uranium-238	1.0	Gastrointestinal Tract
Thorium-234	0.5	Gastrointestinal Tract
Protactinium-234	No data available	
Uranium-234	0.9	Gastrointestinal Tract
Thorium-230	0.05	Bone
Radium-226	0.0004	Bone
Radon-222	Gas	Lung
Lead-210	0.004	Kidney
Bismuth-210	1.0	Gastrointestinal Tract
Polonium-210	0.02	Spleen
Lead-206	Stable	

(Source: Reference 136)

and, indirectly, its daughters, which takes the degree of attachment into consideration:

$$MPC_{air} \text{ (40-hour week)} = \frac{3 \times 10^{-6}}{1 \times 1000 f} \text{ uCi/cm}^3$$

where f = fraction of the equilibrium amount of RaA ions which are unattached to nuclei

Although the ICRP suggests an f = 0.1 for an average atmosphere, measurements in 6 U.S. mines (146) provided f values of between 0.002 and 0.12, with more than 50% of the values being less than 0.03. These latter values would calculate a higher MPC_{air} than the 3×10^{-8} uCi/cm^3 derived from the ICRP calculation (135).

The Working Level method of determining dose is discussed in the following section (3.6.6) and includes comparative information of the WL and ICRP limits.

3.6.6 Measurement of Radon and Radon Daughter Radioactivity

There are many difficulties in the measurement of the radioactivity from the decay of radon (131, 134, 136, 137, 138, 147, 148, 149), but they will not be discussed in detail in this document.

The Manual on Radiological Safety in Uranium and Thorium Mines and Mills, published by the IAEA (135) contains detailed accounts of several methods for monitoring radiation levels in uranium mines.

In Canada, the simple field measurement method of Kusnetz is most commonly employed to quantitate airborne radon daughters in uranium mining atmospheres (135, 148). With this technique, air samples (10 - 15 litres) are drawn through high-efficiency membrane or glass fibre filters for 3 - 5 minutes and, after a delay of 40 - 90 minutes, the alpha activity on the filter paper is measured.

The alpha counter consists of a vial coated with silver-activated zinc sulphide, a phosphor which emits a flash of light when hit by an alpha particle. The "pulses" of light are measured by a photomultiplier tube (photocathode detector) and the number of light flashes emitted is proportional to the alpha-particle concentration present in the sample.

Although it would be preferable if a continuous, personal alpha sampler-detector system were used in mines, this type of instrument has not yet reached a suitable stage of development for field use. Washington (148) and MacLaren (136) presented brief resumes of investigational measurement methods for radon daughters as of 1974 and 1976, respectively.

Instruments which provide an instant readout of radioactivity are available, and are used (136), but will not be elaborated upon here.

The Kusnetz technique uses conversion factors, which are based on decay curves to calculate the radon daughter concentrations in "working levels" (WL).

As currently employed, the method measures radon daughters, and can detect as low as 0.3 WL (± 25%) routinely. The use of scales rather than rate meters can reduce this level to 0.1 WL, but the reliability is only about ± 50%.

In a well-ventilated mine, the radon daughters are seldom in equilibrium with each other or with the parent radon. The health risk from radon and its daughters (which have differing energies) is related to the total absorbed dose and was thought by some experts to be best defined in terms of the total potential alpha energy present, rather than by the concentration in microcuries. Thus, the working level (WL) concept was introduced to assess radon daughter concentrations and provides an alternative to the ICRP approach to calculating the MPC.

One WL is 1.3×10^5 MeV per litre, which is the sum of the alpha-energies released by the decay of the short-lived daughters, RaA, RaB, RaC and RaC', when in equilibrium with 100 pCi of radon (Table 59) and does not take account of the radon present.

One WL is, therefore, the amount of RaA, RaB, RaC and RaC' in any proportion in 1 litre of air which will ultimately emit alpha particles with a cumulative energy of 1.3×10^5 MeV.

The use of the WL "system" of reporting radon daughter levels as potential alpha energy, rather than pCi/l of air, avoids the need to define (or determine) the ratio of each of the daughters present in any particular sample.

TABLE 59

Composition of Working Level at Radon Daughter Equilibrium

Nuclide	**Ultimate alpha-energy per atom (MeV)**	**Total alpha-energy (MeV/100 pCi)**	**Fraction of Total alpha-energy (MeV)**
Radon	Excluded	None	None
RaA	13.68	0.134×10^5	0.10
RaB	7.68	0.659×10^5	0.52
RaC	7.68	0.485×10^5	0.38
RaC'	7.68	0.000×10^5	0.00
		1.278×10^5 $= 1.3 \times 10^5$	1.00

(Source: Reference 135)

Understanding the subject of radon decay and measurement is complicated by the fact, mentioned above, that when radon is first released into the atmosphere it has no daughters associated with it. However, their presence grows rapidly (Figure 79) and, because of their short half-lives, it requires only about 3 hours until the daughters are present in equilibrium with each other. At equilibrium alpha activity from radon and its decay products is three times as much as from radon alone. Therefore, waiting 3 hours to count a sample yields a higher count and eliminates the need to apply a correction factor to account for the time-dependent build-up of radon daughters.

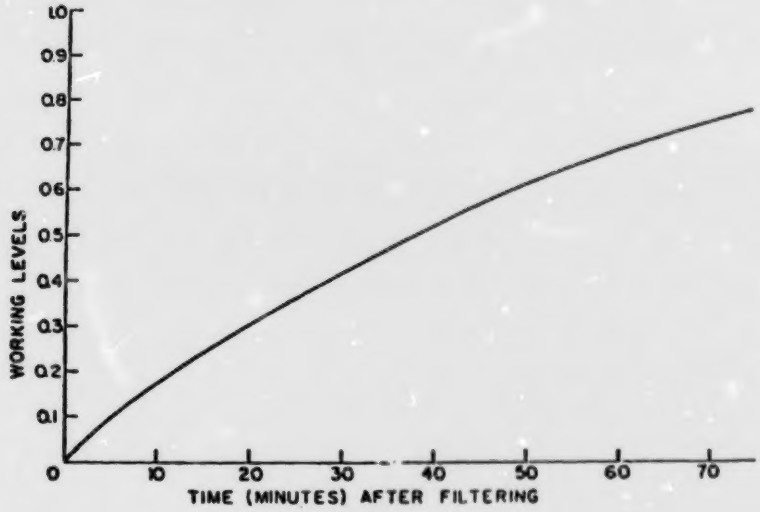


FIGURE 79

Growth of radon daughters in 100 pCi/l of freshly filtered radon

(Source: Reference 150)

It should be emphasized that the Kusnetz assay method measures radon daughters by trapping them on a filter. If radon gas is to be measured it is sampled into a chamber through a filter to remove long-lived alpha-emitting particles, and the chamber sealed. Counting is carried out after a few hours, when the radon and its decay products have reached equilibrium.

Methods are available which permit measurement of the individual concentrations of RaA, RaB and RaC, but will not be detailed in this report (135).

In Canada, the uranium mining regulations stipulate a maximum occupational exposure from radon daughters of no more than 4 WLM (working level months) per year, which is an average of 0.33 WL. It should be remembered that 100 pCi/l of radon in equilibrium with its daughters is 1 WL (and therefore the two MPC's are identical), and that continuous exposure to this concentration gives a dose rate of 12 WLM per year.

Comparing the ICRP MPC of 3×10^{-8} uCi/cm^3 (30 pCi/l) with the 4 WLM limit in force in Canada, it can be calculated that the ICRP limit is about three times more restrictive. This results from the fact that equilibrium concentrations of radon daughters are unlikely to be achieved in a mine and thus 0.3 WL is more likely to be associated with 100 pCi/l of radon than with 30 pCi/l.

The amount of radon present in any location at any given time is determined by the rate of emanation into the atmosphere from the ore body, the rate of removal via the mine ventilation system, and the rate of radioactive decay of the radon gas.

As was briefly pointed out earlier the radon daughter concentration, and therefore the potential hazard, increases rapidly with time for any radon level. Therefore, the higher the ventilation rate in the mine the lower will be the concentration of daughters at any particular time and location. Increasing ventilation reduces the radon hazard in two ways; by diluting the radon concentration and by reducing the time for growth of radon daughters.

One reference (138) recommended that in order to maintain radon-levels below 1 WLM that each pair of men mining ore be provided with no less than 1000 cfm of air discharged from a tube located not more than 300 feet from the face. However, the air supply required will depend on the amount of radioactivity present in a particular mine and such a generalization may not be practical.

As a final note to this section it should be mentioned that, according to the ICRP (135), the WL concept of measuring radiation exposure is of questionable value and is presently being reviewed by an ICRP task group. It is possible that future MPC's may be based on a pulmonary dosimetric model or epidemiological data.

Addendum

Fry (131) provided a lengthy, but useful description of the relationship between radon, its daughters and their emissions. For those interested in an alternative explanation of the WL system his narrative is given below in its entirety:

"Let us consider 100 pCi of radon (that is a quantity giving 222 disintegrations/min) in equilibrium with its short-lived daughters; that is, we have in association with the radon, 100 pCi each of RaA, RaB, RaC and RaC'. We wish to know the total amount of alpha energy emitted during decay of the atoms which go to make up 100 pCi each of RaA, RaB, RaC and RaC'. First we must calculate the number of atoms of each daughter product associated with the activity of 222 disintegrations/min. This is obtained from the relationship that the activity of N atoms is given by N-alpha. Thus for RaA, whose half-life is 3.05 min and whose decay constant, alpha, is

therefore $\frac{0.693}{3.05} = 0.227/\text{min}$, we have

$$N \times 0.227 = 222 \text{ disintegrations/min}$$

whence, $N = 977$ atoms.

Similarly, using the respective half-lives of the other daughter products, we find that there are 8,500 atoms of RaB in 100 pCi of RaB, 6,310 atoms of RaC in 100 pCi of RaC and 0.0009 atoms of RaC' in 100 pCi of RaC'.

Each atom of RaA, when it decays to RaB, gives out 6.00 MeV of alpha energy and this particular RaB atom will eventually become an RaC' atom giving out a 7.69 MeV alpha particle. Thus each of the 977 atoms of RaA is eventually associated with the emission of a total of (6.00 + 7.69) MeV of alpha energy. Similarly, each of the 8,580 atoms of RaB will eventually become an RaC' atom and emit a 7.69 MeV alpha particle. So too will each initial RaC atom, and of course each initial RaC' atom, though there are so few of the latter present that their contribution is literally zero. The total amount of energy eventually emitted from all of the daughter atoms can thus be calculated and summed. This calculation is set out in Table 60 where it can be seen that the short-lived decay products in equilibrium with 100 pCi of radon eventually release a total of 1.3×10^5 MeV of alpha energy. It is interesting to note the seeming paradox that most of the energy -- 90% of it --comes from the two non-alpha-particle-emitting daughters RaB and RaC. This is because of their relatively long half-lives and therefore the correspondingly larger number of atoms of each that must be present to give 100 pCi of activity.

TABLE 60

Relevant Data Used to Derive The Working Level

Nuclide	Alpha energy (MeV)*	Half-life	No. atoms in 100 pCi	Ultimate alpha energy/atom	Total ultimate alpha energy (MeV/100 pCi)
RaA	6.00	3.05 m	977	6.00 + 7.69	0.134×10^5
RaB	0.00	26.8 m	8580	7.69	0.660×10^5
RaC	0.00	19.7 m	6310	7.69	0.485×10^5
RaC'	7.69	2.7×10^{-6}m	0.0009	7.69	0.000×10^5
				Total	1.279×10^5
				Round up to	1.3×10^5

(*) MeV = million electron volts

The assumption is now made; any combination of RaA, RaB, RaC and RaC',–regardless of its state of radioactive equilibrium, which will result in an ultimate emission of 1.3×10^5 MeV of alpha energy has the same potential hazard as the equilibrium combination, if distributed in the same volume of air. If the volume of air is taken as one litre, a new unit, the "Working Level" (WL) emerges.

Thus, one WL is defined as 'any combination of the short-lived decay products of radon (RaA, RaB, RaC and RaC') in 1 litre of air which will result in the ultimate emission by them of 1.3×10^5 MeV of alpha particle energy.'

Note that 1 WL is equivalent to 100 pCi/l of radon only in the one special case when the radon is in equilibrium with its short-lived decay products. To express a radon concentration, measured in pCi/l, in working levels, it is necessary to know the state of disequilibrium between the radon and its short-lived daughters under the conditions pertaining at the time.

Thus we can write that, in general,

F x (Concentration in WL = Concentration in pCi/l)/100

where F is a factor, variously called the 'disequilibrium factor' or 'working level ratio', which is a measure of the lack of equilibrium between the radon and its short-lived daughters. F is very variable in practice and rarely has a value of one. Because of the half-lives of RaB and RaC it would take about three hours for initially-fresh radon to come into equilibrium with its short-lived daughters. Any process which removes the daughter products, particularly movement and dilution of the air, will ensure that F remains less than one. In a space where the ventilation rate was as low as one air change/hour, F would be kept to about 0.7 and would drop to about 0.4 at two air changes/hour.

A WL is a measure of a concentration. To obtain an accumulated to integrated exposure, one must multiply by the time of exposure. Integrated exposures are usually measured in 'Working Level Months' (WLM). Breathing air with a concentration of 1 WL of radon daughters for the working hours in a month (taken as 170 h) results in an exposure of 1 WLM.

Finally, a unit for rate of exposure is required. A person who accumulated 1 WLM of exposure each month would be exposed at the rate of 12 WLM/a. The unit WLM/a is commonly used to measure exposure rate. An exposure rate of 4 WLM/a is important in what follows. This exposure rate will result from working continuously for 170 h/month in an atmosphere from a daughter prdouct concentration of 0.33 WL" (131).

3.6.7 Thoron and Its Daughter Products

Earlier mention was made of thoron, which is a radioactive gas similar to radon and found in association with uranium. Thoron sources emit alpha particles and are a health hazard, but it is thought that the hazard from thoron is only about 1/10th of that from radon. In general, it appears that in Canadian mines thoron is present at least an order of magnitude less than that for radon and, therefore, it is not thought to constitute a significant health problem in Canadian uranium mines. This point should be confirmed, however, as thorium is insoluble and becomes fixed in bone in a similar way to plutonium. It is eliminated slowly and therefore could be hazardous.

In any case, adequate control of radon levels should reduce thoron concentrations to acceptable concentrations. The MPC in air for radiation workers is 3×10^{-7} uCi/cm^3, with the ThB ($t_{\frac{1}{2}}$ 10.6 hours) considered the prime contributor to any possible hazard.

3.7 Regulation of Diesel Use in Mines

Important to any recommendations concerning the use of diesels underground is a knowledge of current regulations in force in Canada, as well as other countries. A summary of relative regulations and recommendations is given in Table 61.

As can be seen by perusing Table 61, the regulations vary, not only from country to country, but within countries. However, the majority are based on ACGIH-TLV values. This information is provided for comparative purposes only, and no discussion of the differences will be undertaken.

TABLE 61: Regulation of Diesel Use in Mines

COUNTRY		AUSTRALIA	WESTERN AUSTRALIA (64)	NEW SOUTH WALES (64)	TASMANIA (64, 152)	BRITAIN (12, 64)
VENTILATION REQUIREMENTS			- 3.8 m^3/kw with minimum of 1.9 m^3/kw, at least 141 m^3/min. (150 cfm/bhp) for each diesel vehicle	- as for West. Australia but 141 m^3/min. for each vehicle or 5000 cfm, whichever is the greater	- as specified by inspector - determine CO, CO_2, NO_x, aldehyde at 3 modes for each engine	- recommended 3.47 m^3/min. bhp or 150 cfm/bhp
					- calculate requirements on "worst case" basis	
FUEL			Not more than 0.5% S	Not more than 0.3% S	Not more than 0.5% S	Not more than 1.0% S
MAXIMUM EXHAUST EMISSIONS REQUIREMENTS (ppm)	CO	2500		1500		1500 - at idling and full load
	NO_x	2000		1000		1000
	NO_2					
	OTHER					
MINE ATMOSPHERE REGULATIONS (ppm)	CO_2	5000			2500	
	CO	50			50	50
	NO					
	NO_2	5				
	NO_x				12.5	
	SO_2	5				
	H_2S	10				
	ALDEHYDES	5 (HCHO)			5	
	DUST					
	OTHER					
MISCELLANEOUS AND NOTES					- use additive ACGIH method for mixtures - cannot use catalytic converters - water scrubbers - prohibit excess of black smoke	- if mine CO ≥ 100 ppm shut down until < 50 ppm - use ACGIH standards - no idling permitted - at least 15:1 exhaust dilution before discharge

TABLE 61 (Cont'd)

COUNTRY		CANADA			FRANCE (64)	WEST GERMANY (64)
		ONTARIO (153)	SASKATCHEWAN (154)	NOVA SCOTIA (7)		
VENTILATION REQUIREMENTS		- Not less than 100 cfm/bhp	- Not less than 100 cfm/bhp		- calculated from engine NO_x/CO and $d = \frac{NOx}{10} + \frac{CO}{50}$ E (vol. exhaust) = $\frac{1}{2} \cdot \frac{n}{60}$ c n = rpm c = cyl. vol. - k = time operating in mine/total time to get k (presence factor) - ventilation requirement = k D E or 3.8 m^3/sec for a Deutz F8B-714 and k = 0.8	- 2.5 m^3/min. bhp (non-gassy mine) - CO sets requirement (see notes)
FUEL		- Not more than 0.5% S - Not less than 125°F FP	- Not more than 0.5% S - Not less than 125°F FP			
MAXIMUM EXHAUST EMISSIONS REQUIREMENTS (ppm)	CO	1500	1500 (before scrubber)			500
	NO_x					750
	NO_2					75
	OTHER					
MINE ATMOSPHERE REGULATIONS (ppm)	CO_2		5000	2000 (coal) 2500 (other)		
	CO	20 (weekly)	24		50	- sets vent. reqmt. (see notes)
	NO					
	NO_2	5	2			
	NO_x				10	
	SO_2					
	H_2S					
	ALDEHYDES					
	DUST		- ACGIH nuisance dust standard			
	OTHER		- Not less than 19% O_2			
MISCELLANEOUS AND NOTES		- meet ACGIH standards - instruct operators on undue idling - no regulation	- only permit "short" time idling		- must insure $\frac{CO}{50} + \frac{NO}{25} + \frac{NO_2}{5} \leqslant 1$ - 2.4 m^3/bhp meet above for Deutz	- 20:1 dilution of exhaust before discharge - CO sets ventilation requirements: (600 ppm - 3 m^3/bhp 600 - 800 ppm - 4 m^3/bhp 800 - 1200 ppm - 6 m^3/bhp

TABLE 61 (Cont'd)

COUNTRY		HUNGARY	JAPAN (64)	ROMANIA (64)	SOUTH AFRICA	SWEDEN (64)	SWITZERLAND (64)	U.S.A.	VENEZUELA
VENTILATION REQUIREMENTS			- 3 m³/kw	- CO sets ventilation 500 ppm - 3 m³/min. bhp 500-800 ppm - 4 m³/min. bhp 800-1200 ppm - 6 m³/min. bhp	- Not less than 123 cfm/bhp (8 m³/s per 100 hr)	- Bolidan Formula - Q = 0.27 PKS/3600 or 2500 m³/kg fuel burned	$\frac{NO}{25}+\frac{NO_2}{5}+\frac{SO_2}{5}\leq 0.8$ and rest NMT TLV - average reqmt. 5 m³/min. bhp - with S-free fuel 4 m³/min. bhp		- has no regulations relative to diesel use
FUEL						- Not more than 0.1% S		- Not more than 0.5% S	
MAXIMUM EXHAUST EMISSIONS REQUIREMENTS (ppm)	CO			500 (new); 1200 (old)	2000	600			
	NO_x				1000				
	NO_2								
	OTHER			- 80 mg/m³ particulate (new) - 150 mg/m³ particulate (old)		- NMT 30 mg/m³ particulate (no load/ 800 rpm) - NMT 80 mg/m³ (full load)			
MINE ATMOSPHERE REGULATIONS (ppm)	CO_2	1000			5000	1500 (30 min.) 500 (8 hr.)	5000	5000	
	CO	50		- sets. vent. reqmt. (see above)	100	50 (30 min.) 25 (8 hr.)	50	50	
	NO	5						25	
	NO_2					3 (30 min.) 2 (8 hr.)		5	
	NO_x				5	30 (30 min.) 20 (8 hr.)			
	SO_2	10						5	
	H_2S	10			20				
	ALDEHYDES								
	DUST				- as specified by investigator	2.0 mg/m³ respirable (coal dust)			
	OTHER					2 O_2 19% (30 min.) O_2 20% (8 hr.)			
MISCELLANEOUS AND NOTES			-diesels not permitted in coal mines		- no idling permitted; must have water scrubber	- ACGIH stnds. for workplace but lower for mines - no idle or turbocharged engines permitted	- hp calculated by x actual fuel used x specific fuel consumption (180 g/bhp-hr)	-must dilute exhaust prior to discharge -exhaust component requiring greatest dilution sets vent. requirements	

3.8 Canadian National Air Quality Objectives

In late 1976 Fisheries and Environment Canada published the results of deliberations by a Federal-Provincial Committee on Air Pollution (151) entitled "Criteria for National Air Quality Objectives".

It is relevant to summarize this publication for comparison with mine atmosphere pollutant levels. The significance of this is enhanced when it is recalled that a miner spends 1/3 of each working day in the mining environment. That is, approximately (37.5/168 x 100) 22% of his weekly intake of air is in the mine. This is a significant portion of his total exposure, and therefore the ideal situation would be where the mining atmosphere approximated ambient air quality.

In instances where the air quality in a mine cannot be maintained comparable to the ambient air, the present authors feel that the worker should be so informed and his health risk estimated so that he can make an informed judgement regarding his choice of employment.

Table 62, taken from reference 151, provides a summary of recommended national air quality objectives. Some discussion and interpretation of these values is in order to place them in perspective.

Sulphur Dioxide

Various reports have suggested that 0.1 - 0.2 ppm 24-hour average values lead to increased absence from work, hospitalization and illness, particularly in those with respiratory diseases. These effects occurred in the presence of other pollutants, including suspended particulates.

Suspended Particulates

This term generally refers to chemically inert particles under 100 u in diameter. Some reports suggest a health effect when the annual average exceeds 130 ug/m^3. However, this is always assumed to be in the presence of SO_2. In the particular instance cited, there was a reported increased frequency and severity of lower respiratory disease among children where both the annual smoke level and SO_2 exceeded 130 ug/m^3.

Carbon Monoxide

Based on various published works, including studies which demonstrated potentially adverse effects in patients with coronary artery disease,

TABLE 62

Sumary of Recommended National Air Quality Objectives

		Air contaminant									
		Sulphur dioxide ($\mu g/m^3$)	(ppm)	Suspended particulates ($\mu g/m^3$)	Carbon monoxide (mg/m^3)	(ppm)	Oxidants (ozone) ($\mu g/m^3$)	(ppm)	Nitrogen dioxide ($\mu g/m^3$)	(ppm)
Maximum acceptable limit	1 h	900	0.34		35	30	160	0.08	400	0.21
	8 h				15	13				
	24 h	300	0.11	120			50	0.025	200	0.11
	1 yr	60	0.02	70			30	0.015	100	0.05
Maximum desirable limit	1 h	450	0.17		15	13	100	0.05		
	8 h				6	5				
	24 h	150	0.06				30	0.015		
	1 yr	30	0.01	60			20	0.01	60	0.03

Conversion factors used to convert mass units to volume units of concentration (at 25°C and 760 mm Hg).

Sulphur dioxide:	3.82×10^{-4}
Carbon monoxide:	0.87
Oxidants (ozone):	5.1×10^{-4}
Nitrogen dioxide:	5.32×10^{-4}

(Source: Reference 151)

saturation levels of carboxyhemoglobin (COHb) of 5% and over may be physiologically important. The criteria discussion pointed out that effects at levels of COHb betwen 2 and 4% were not well established and no effects have been noted at concentrations of less than 2%.

The ambient air quality standards were set so that COHb levels would not exceed 5% in non-smokers. A safety factor was recommended so that COHb level in non-smokers would remain below 3%.

The CO limit was calculated using a modification of the equation of Larsen (151):

$$C_{COHb} = a\, C_{CO}\, t + E$$

where C_{COHB} = degree of COHb saturation

a = activity factor (i) = 0.018 under conditions at rest with a tidal volume at 6 l minute

(ii) = 0.048 under conditions of light to moderate work with a tidal volume of 18 l/minute

(iii) = 0.1 under conditions of heavy work (arbitrarily selected)

C_{CO} = concentration of CO in air in ppm

t = length of exposure in hours with maximum of 12 hours at which time equilibrium conditions are assumed to be operative

E = 0.5% COHb representing endogenous CO production

The amount of CO measurable in relatively unpolluted air ranges between 0.029 -1.15 mg/m^3. Endogenously-produced CO results in a biological background level of 0.42 ml/hour and in COHb levels of about 0.5%.

Nitrogen Dioxide

The standard for NO_2 was set primarily on the basis of an epidemiological study carried out in the Chattanooga area. Ventilatory performance was lower and incidence of acute respiratory conditions greater in children in high NO_2 exposure locations. The 24-hour NO_2 levels, over 6 months, were between 117 and 205 ug/m^3.

Perception limits (415 ug/m^3) were also considered when setting the 1 hour-400 ug/m^3 maximum acceptable standard. This value is about one-tenth the level at which an increase in both inspiratory and expiratory flow resistance occurred after exposure in the presence of an equal concentration of SO_2. The 100 ug/m^3, 1 year average is a concentration slightly below the ranges of NO_2 found to have adverse long-term health effects in the Chattanooga study.

A table of example of NO_x levels in Canadian cities is included (Table 63).

3.9 Mines Atmospheres

3.9.1 Measurement Methods for Environmental Pollutants

In a laboratory and field study carried out by J. Johnson et al (43, 44) for MESA, various instruments were evaluated for their applicability to measuring contaminants in mine atmospheres. These reports contained extensive quantitative and qualitative assessments of the available analytical instrumentation and the reader is referred to them for full details.

TABLE 63

Observed Levels of Nitrogen Oxides (NO_x) in Canadian Cities

City	Year	Mean Annual Concentration (ug/m^3)	Peak Concentration (ug/m^3)	Estimated Mean Annual NO_2 Concentration (ug/m^3)
Windsor	1969	38	282	15 - 33
	1970	75		30 - 45
Toronto	1969	66	526	26 - 40
	1970	113		45 - 68
Edmonton	1968	24	1436	10 - 14
	1969	24	1021	10 - 14
Westmount*	1969			75
Montreal*	1969			70
	1971			26
	1972			26

Note: NO_x values are expressed in terms of NO_2

* The levels are those for nitrogen dioxide only

(Source: Reference 151)

In general, instrumentation appears to be available which can adequately measure the gaseous and particulate substances found in mine atmospheres. In summary Johnson et al found the instruments in Table 64 acceptable, although modification and frequent adjustments were necessary for many of them. More details can be found in references 43 and 44.

It is imperative that industry and regulatory bodies agree on the details of mine air sampling and contaminant measurement methods to be used, so that they both work towards compliance with existing standards using the same armamentarium. Reports, such as that in the Toronto Globe and Mail of February 6, 1978, in which significant differences in radon concentrations (the mine values were 30% below those of the Ministry of Labour) were tabled, are of serious concern

TABLE 64

Listing of Analytical Instruments in MAML

BENDIX 8101-B NITROGEN OXIDES ANALYZER
"Chemiluminescent"; NO, NO_2 and NO_X in ranges of 0 - 20, 50, 100, 200 and 500 ppm full scale.

BENDIX 8501-5CA CARBON DIOXIDE ANALYZER
"NDIR", CO_2 in ranges of 0 - 0.5, 2 and 5% full scale

BENDIX 8501-5CA CARBON MONOXIDE ANALYZER
"NDIR", CO in ranges of 0 - 50, 250, 500 and 2500 ppm full scale

BENDIX 8851 DYNAMIC CALIBRATION SYSTEM
Used for calibration of Bendix 8101-B nitrogen oxides analyzer. NO from bottle span gas and NO_2 from permeation tube or ozone reaction with NO. Zero and dilution air obtained from a BENDIX 8832 AIR CLEAN-UP SYSTEM.

THERMO-SYSTEMS 3030 ELECTRICAL AEROSOL SIZE ANALYZER
"Electrical charging and mobility analysis", measures particulate matter by number/volume in 10 size increments between 0.0032 and 1.0 um.

(Source: References 43 and 44)

and totally unnecessary. Although this particular instance may be the exception rather than the rule, it is nonetheless disconcerting and may sound warning of a greater prevalence of such situations than has been previously thought to exist.

Recently, the Michigan Technological University group published an up-date of instrumentation used in monitoring mine air quality (155). Details were discussed in section 3.3.2

3.9.2 The Conversion of NO to NO_2 In the Mine Atmosphere

The toxicity of NO_2 (TLV of 5 ppm) is greater than NO (TLV of 25 ppm), and thus there is concern over the rate and extent of conversion of NO to NO_2 ($2NO + O_2 \longrightarrow 2NO_2$) in the underground environment, which could result in insidious and dangerous concentrations of NO_2. That this reaction occurs has been adequately documented (54, 66, 156, 157, 158). It seem sappropriate to discuss this potentially-hazardous occurrence prior to a more detailed account of mine pollutant levels.

Harkins and Goodwine (156) determined that the rate of conversion of NO to NO_2, which occurs in a relatively static atmosphere, could be predicted using the equation:

$$-\left(\frac{d(NO_2)}{dt}\right) = k(NO)^2(O_2)$$

The following table of conversion values was calculated using the above formula:

NO (ppm)	NO_2 Formation (ppm/minute)
1000	210
100	2.1
10	0.021
1	0.0021

Therefore, at high NO concentrations the rate of conversion is relatively rapid compared to the rate at lower concentrations. A table from the Matheson Gas Data Book exemplifies this point, as follows:

NO Concentration (ppm)	Time to Convert to NO_2 (%)		
	25%	50%	90%
1000	1.4 min.	4 min.	3.6 min.
100	14 min.	40 min.	360 min.
10	2.3 hrs.	7 hrs.	63 hrs.
1	24 hrs.	72 hrs.	648 hrs.

Dainty's group (56) calculated that, at equilibrium, the conversion rate for an atmospheric NO concentration of 14.5 ppm would be 0.044 ppm/minute. At this initial concentration the conversion of NO to NO_2 would result in an exit concentration of 1 TLV equivalent of nitrogen oxides after a one-hour residence time.

It was calculated that should an idling engine emit 400 ppm of NO in its undiluted exhaust for one minute, which could result in the formation of 34 ppm NO_2, there could be a 34% increase in the equivalent NO concentration to 536 ppm.

Fine (157) expanded the equation of Harkin and Goodwine so that the rate of conversion could be accurately predicted. Using the Fine calculation Stewart et al (67) computed the conversion of NO to

NO_2 for various times and initial NO levels. Using the suggested ACGIH additive toxicity equation for NO and NO_2 ((NO)/25 + (NO_2)/5), the data were graphically plotted against a "toxicity index" as shown in Figure 80. The number '1' on the vertical (ordinate) axis represents the TLV for any mixture. Thus, the increase in toxicity of the nitric oxides with time is readily apparent. In particular, it is obvious that the toxicity of NO_X's would rapidly become serious at a starting NO concentration of 15 - 25 ppm. It must be remembered, however, that this series of events occurs as indicated in a 'static' atmosphere.

It is clear from the above discussion that rapid dilution and removal of the exhaust is required to reduce the conversion of NO to NO_2.

Although no data were found which provided an indication of the importance of acid production in the mine atmosphere, the report of work such as that of Greig and Hall (159) should be kept in mind. They showed that the removal of NO_2 increased in the presence of water vapour (and mines often have a relative humidity of 80 - 100%) as a result of the reaction between NO_2 and adsorbed water in the following fashion,

$$2NO_2\ (\text{gas}) + H_2O\ (\text{adsorbed}) \longrightarrow HNO_3\ (\text{adsorbed}) + HNO_2\ (\text{gas})$$

thus forming adsorbed oxyacid. The TLV for HNO_3 is 2 ppm and, therefore, if this reaction were to occur to a significant extent it could result in an undesirable acid level in the atmosphere. Again, however, dilution of the exhaust gas would reduce the significance of this reaction due to suppression of formation of NO_2, as well as diluting whatever NO_2 might be formed.

Average NO to NO_2 conversion rates were measured over a 1-hour period by A. D. Little during their coal mine diesel pollutant study (160). This was done by measuring the change in NO/NO_2 levels upstream and downstream. The average data are given in Table 65.

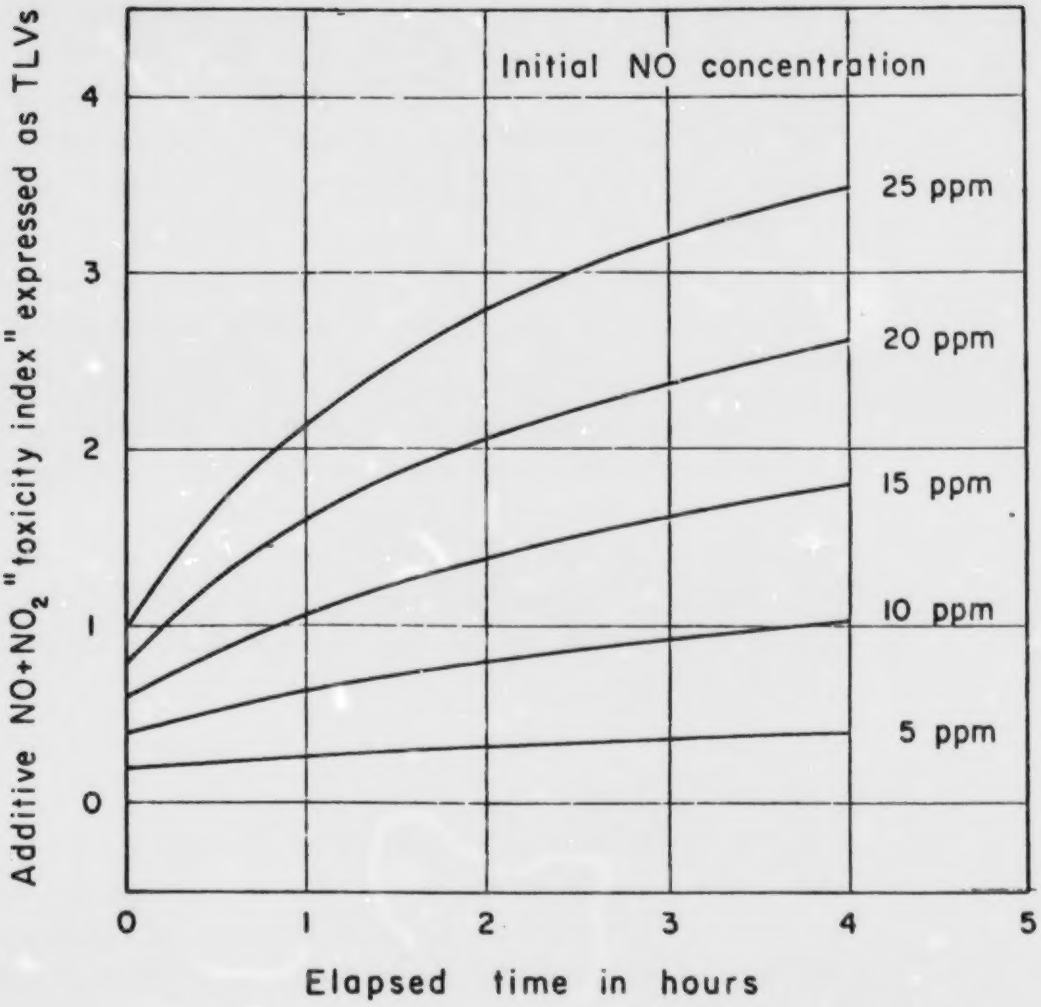


FIGURE 80

The additive NO + NO_2 "Toxicity Index" change with elapsed time due to NO and NO_2 conversion

(Source: Reference 67)

TABLE 65

NO - NO_2 Conversion Data

Gas	Average Upstream Concentration (ppm)	Average Downstream Concentration (ppm)
NO_2	0.15	0.15
NO	1.7	2.1
CO	4.2	4.6

It was concluded that in the mining atmosphere studied, NO was not converting to NO_2 at a hazardous rate.

3.9.3 Sampling and Measuring of Atmospheric Pollutants

Two basic approaches are taken in sampling mine atmospheres:

1. "Grab" or single samples or short-time samples,
2. Continuous samples.

Grab or random samples are, as the name implies, taken at a given point in time and usually include a relatively short sampling period, in the order of a few minutes. Grab samples will only detect a high level of a pollutant if it is present at the time of sampling. If it is desired to determine whether a work atmosphere is in compliance over an 8-hour work shift, then many random samples would have to be taken during the 8 hours. Even then the statistical reliability is not particularly good, and concentrations of contaminants 25 - 70% above the TLV are usually needed to detect non-compliance with standards (44).

For monitoring exhaust concentrations during a particular engine operating mode, grab sampling is acceptable, although continuous monitoring is better.

Sampling using Drager or similar tubes, relying on visual colour indicators to assess pollutant levels constitutes a grab sample. It should be emphasized that although Drager tube-type methods are simple and inexpensive, they have an accuracy of about ± 25% (44, 161). According to Johnson and colleagues (44) the tubes gave higher readings than for comparable samples measured with more accurate instruments. Whether this was due to some interfering substance particular to the mine studied, or was a general characteristic of the tubes was not known.

Continuous atmosphere sampling or monitoring is the preferred technique, and permits time-weighted average data to be obtained for a full or partial workshift. With continuous sampling, an instrument is used which continuously samples, measures and records, or plots pollutant concentrations. Alarm systems can be integrated into continuous monitoring equipment which can permit quick decisions and corrective actions to be made.

For monitoring mine atmospheres the preferred method would be to mount continuous sampling-analyzer equipment on each LHD machine. This would provide a record of pollutant levels throughout work cycles and zones in the mine most likely to have the highest levels of diesel exhaust. This is the approach taken by Johnson's group (9) in monitoring mines atmospheres with their mobile laboratory.

The outline of analytical instruments available, and presented in Section 3.3.2, is applicable to environmental monitoring, and will not be repeated here. Additional information on analytical methods can be found in the coal mine pollutant survey published by A. D. Little (160).

For a review of sampling and other problems related to quantitating the underground atmosphere the reader is referred to a recent presentation by Bardswich (161).

3.9.4 Mine Air Diesel Pollutant Levels

The present authors neither carried out analytical work nor were provided with official mine atmosphere survey data. Therefore, the estimates used in the following discussions had to be made on data retrieved from the published literature or provided by mining companies. The applicability and accuracy of the information discussed in this section must be assessed keeping this point in mind.

A review of a few of the more descriptive reports on pollution levels in dieselized mines and tunnels will demonstrate the degree of variability which has been found, but they nevertheless provide a basis for making average estimates relevant to "typical" mining atmospheres.

Sutton (72) presented environmental data for seven U.S. underground mines using diesel-powered equipment. The author concluded that, "although individual spot samples for CO and CO_2 indicated elevated peak concentrations, the time-weighted averages were below the current TLV of 50 ppm and also suggested the lower value of 35 ppm". Gravimetric, respirable dust (which included both diesel soot and rock dust) did, on occasion, exceed the allowable levels. In one instance it ranged to as high as 12.9 mg/m^3. Less than 10% of the NO_X was comprised of NO_2. It was estimated that diesel duty cycles were in the range of 20 to 50%.

The A. D. Little Company completed a project for the U.S. Bureau of Mines on the effect of diesel emissions on the quality of a coal mine atmosphere in Martin County, Kentucky (160). The publication dealt in detail with the analytical methods, their shortcomings and advantages. A summary of the results is given in Table 66. It should be noted that the particular mine surveyed used mainly Caterpillar diesels in their heavy-duty machines.

By comparing the atmospheric data with tail-pipe exhaust analysis, an average dilution of about 100-fold was calculated (160).

The breathing zone concentrations of contaminants in 22 dieselized metal and non-metal, and 2 coal mines were published in 1977 by LFE Corporation in a report of a study conducted for the U.S. Bureau of Mines (162). In addition to the usual gaseous diesel emissions, such as CO, NO, NO_X, CO_2, aldehydes, etc., polynuclear aromatic (PNA) levels were also investigated, using GLC methods. The sensitivity of the method was indicated to be 0.1 ug of PNA. Sampling for PNA's was carried out using a silver membrane filter followed by extraction of PNA's with methylene chloride and injection into a GLC with a flame ionization detector. Thus, only PNA's associated with particulate were analyzed.

TABLE 66

Pollutant Levels in the Martin County Coal Mine Compared to TLV's

Species	Concentration Units	Measured Concentrations Lowest	Mean	Highest	Threshold Limit Value
Aldehydes	mg/m^3	0.004	0.045	0.19	3* - formaldehyde 180 - acetaldehyde 0.25 - acrolein
Aromatic Hydrocarbons	mg/m^3	0.13	0.69	2.1	50 - napthalene
Carbon Dioxide	ppm	450	650	1150	5000
Carbon Monoxide	ppm	1.5	7.6	14	50
Formaldehyde	mg/m^3	0.007	0.038	0.059	3*
Nitric Oxide**	ppm	1	3	5	25
Nitrogen Dioxide	ppm	0.10	0.34	0.47	5
Oxygenates	mg/m^3	0.006	0.039	0.11	19 - phenol 22 - cresol (all isomers)

(*) Ceiling Value

(**) Estimated from the average NO/NO_2 ratio (10 to 1) obtained with direct reading instruments.

(Source: Reference 160)

The results, summarized in Table 67, indicated the workers were usually exposed to levels of exhaust components of less than 50% of the TLV's.

Of the PNA's sought, only phenanthrene (10 ug/m^3 average) and anthracene (1 ug/m^3) were detected. The report concluded that formaldehyde, NO_2, CO and CO_2 were the most appropriate contaminants for indicating hazardous concentrations of diesel emissions.

The LFE surveys indicated that an average air velocity of at least 150 cfm should provide adequate ventilation when working in areas where diesels operate. Details of the types of diesel engines used were not given, so it is not known how this data might relate to the Canadian scene.

Sutter (73) reported the results of a comprehensive study of the air quality resulting from diesel operation during tunnel construction. The average exposure of the tunnel workers was:

NO	1.5 - 3.5 ppm
NO_2	Less than 0.5 ppm
CO	Less than 10 ppm
CO_2	Less than 1000 ppm
SO_2	approx/equal to 0.02 ppm
HC as methane	5 - 20 ppm
diesel soot	0.5 - 1.5 mg/m^3
mineral dust	0.5 - 1.5 mg/m^3 (10% respirable)

Nitrogen dioxide was less than 10% of the NO levels and frequently was so low as to be undetectable. An interesting finding was that operators of diesel equipment at the face or heading (loaders and excavators) of the tunnel were exposed to levels about twice the concentration of contaminants in the general tunnel air. Operators of moving equipment, such as haulage vehicles and graders, were exposed to the pollutant levels of the main tunnel air. The equipment was found to operate about 25 - 30% of the time at an average load of 50%. Therefore, only 13 - 15% of the total diesel capacity was used. Because ventilation was installed based on total hp in the tunnel the

TABLE 67

Summary of Analytical Results of Phase II Samples

Contaminant	Type of Sample	No. of Samples	Mean (ppm)	Range (ppm)	TLV (ppm)
Total Dust	Source	8	50.2 mg/m^3	0.5-236 mg/m^3	--
	Breathing zone and area	13	4.6 mg/m^3	0.2-14 mg/m^3	10 mg/m^3
Acrolein	Source	7	0.9	0.06-1.4	--
	Breathing zone and area	16	0.172	0.001- 2	0.1
Formaldehyde	Source	10	5.9	0-35	--
	Breathing zone and area	23	0.7	0-6.6	C 2
NO_2	Source	5	8.4	0.5-20	--
	Breathing zone and area	29	0.8	0-5.5	C5
CO_2	Source	5	15,400	4,000-25,000	--
	Breathing zone and area	23	1,600	145-5,000	5,000
CO	Source	6	122	10-300	--
	Breathing zone and area	21	12.4	0-23	50
SO_3	Source	4	3.2	0.06-11.5	--
	Breathing zone and area	9	0.07	0.001-0.5	0.2
NO	Source	3	5.1	0.1-13.4	--
	Breathing zone and area	10	10.3	0.4-57	25
SO_2	Source	5	0.1	0-<0.5	--
	Breathing zone and area	6	0.8	0-5	5
Phenanthrene	Source	8	0.001 mg/m^3	0-0.008 mg/m^3	--
	Breathing zone and area	13	0.01 mg/m^3	0-0.17 mg/m^3	--

TABLE 67 (Cont'd)

Summary of Analytical Results of Phase II Samples

Contaminant	Type of Sample	No. of Samples	Mean (ppm)	Range (ppm)	TLV (ppm)
Anthracene	Source	8	0.05 mg/m^3	0.02-0.2 mg/m^3	--
	Breathing zone and area	13	0.01 mg/m^3	0.000050-0.004mg/m^3	--
Pyrene	Source	8	<0.008 mg/m^3	0.004-<0.02 mg/m^3	--
	Breathing zone and area	13	<0.002 mg/m^3	0.00006-<0.0006mg/m^3	--
1,2 Benzo-anthracene	Source	8	<0.02 mg/m^3	0.0004-<0.004mg/m^3	--
	Breathing zone and area	13	<0.005 mg/m^3	0.0001-<0.008mg/m^3	--
Chrysene	Source	8	<0.05 mg/m^3	0.005-<0.2 mg/m^3	--
	Breathing zone and area	13	<0.005 mg/m^3	0.0002-<0.002 mg/m^3	--
Benzo(e)-	Source	8	<0.11 mg/m^3	0.04-<0.4 mg/m^3	--
	Breathing zone and area	13	<0.001 mg/m^3	0.0003-<0.001 mg/m^3	--
Benzo(a)-pyrene	Source	8	<0.17 mg/m^3	0.06-<0.6 mg/m^3	--
	Breathing zone and area	13	<0.002 mg/m^3	0.008-<0.005 mg/m^3	--

(Source: Reference 162)

reduced load factor meant that the tunnel was "overventilated". The author pointed out that they used the fractional maximal allowable - additive approach (similar to that recommended by the ACGIH), is calculating ventilation needs, as follows:

$BNO + BNO_2 + BSO_2$ 0.8, where Conc./TLV = B

In a 1977 communique from the French Laboratoire du Centre D'Etudes et Recherches des Charbonnages de France (163), atmospheric data were provided for an underground coal mine site where Deutz-equipped trucks operated.

Continuous monitoring of CO, NO and NO_2 was carried out at various locations in the workings. Table 68 provides average NO concentrations in one workroom and at the mine face where one vehicle was in operation.

TABLE 68

Average NO Concentrations

Day	NO(ppm) Mine Face	NO(ppm) In Work Room
1	3.5	5.5
2	2.0	5.5
3	3.5	8.0

(Source: Reference 163)

Table 69 gives the mean CO, NO and NO_2 levels in the workroom and in the driver's compartments, along with the limits as calculated from the additive formula:

$$\frac{CO}{50} + \frac{NO}{25} + \frac{NO_2}{5}$$

TABLE 69

Average Contaminant Concentrations

		Concentration of Pollutant (ppm)							
		CO		NO_2		NO		Additive Formula	
Day	Room	Room	Truck	Room	Truck	Room	Truck	Room	Truck
1	K1	4.0	4.5	0.3	0.3	5.5	7.5	0.3	0.46
2	K1	6.5	6.0	0.2	0.1	8.0	7.0	0.48	0.43
3	K1	4.0	4.0	0.1	0.3	7.0	7.0	0.38	0.44
3	K2	5.5	4.5	0.2	0.3	7.0	6.0	0.44	0.41

(Source: Reference 163)

The data strongly indicate that the driver's exposure was basically the same as found in the general room atmosphere and that, in both instances, the ventilation kept the pollutant levels well under control.

It was found that the level of contaminants generally paralleled the activity of the trucks, with exhaust gas concentrations increasing at higher work loads.

It was estimated that the air into K1 was equivalent to 50 - 60 l/hp.sec., and into room K2, 40 l/hp.sec. K1 was ventilated with fresh air at a rate of 4 - 5 m^3/s and K2 at 3 - 4 m^3/s.

During continuous operation the average concentration over a 2 - 3 hour period for CO was 5 - 10 ppm, for NO 10 ppm and for NO_2 0.4 ppm. The value of the additive formula was 0.6, which was considerably below the maximum permissible value of 1.0.

In this study ventilation requirements appeared to be best estimated on the basis of NO concentrations. It was noted that NO_2 and CO were rarely as high as their maximum acceptable concentrations, which were equal to the MAC (5)/10 = 0.5 for NO_2, 50/5 = 10 ppm for CO. The value of MAC(25)/2 = 12.5 ppm for NO was exceeded on several occasions.

Interestingly, one truck was almost new and the other had worked in the mine for 3000 hours, yet there were no significant differences in their exhaust pollutant outputs.

One of the most extensive, on-going investigations on the contribution of diesel exhaust to the mining environment has been carried out at the White Pine Copper mine in Michigan. This operation uses the room and pillar method of mining, and is heavily dieselized. It is comparable to Canadian metal mines such as INCO in that about 75% of the LHD and haulage diesel power is provided by Deutz engines (43, 44) (Table 70). Details of analytical instruments used, including both the vehicle mounted and those in a mobile laboratory constructed specifically for the project, can be reviewed in the original publications (43, 44).

TABLE 70: Data on Diesel Equipment Used in the White Pine Mine for Ventilation Requirements

Vehicle	Bureau of Mines approval number	Engine type and horsepower	Maximum fuel rate, lb/hr (1)	Ventilation required at max. fuel rate, cfm	Typical fuel rate in mine lb/hr	Typical hours use per shift per vehicle	Number of vehicle usages per units per shift
Wagner scoop tram	24-108	Deutz FBL714 174	70	20,000	21.1	6.5	1.67
Wagner ore car	24-55	Deutz F12L714 266	107	30,000	24.1	6.5	1.50
Wagner endloader scoopmobile	24-67	Deutz F6L714 130	52	15,000	17.3	6.5	0.50
Personnel carriers	--	GMC-Detroit #453 32	--	--	--	0.25	2.00
Ensign CR100	--	Ford DD 172 59	22.6	9,400	--		
Willys jeep	--	Perkins 38	--	--	--	0.45 (All 3 combined)	3.00
Willys jeep	24-79	Ford DD 172 59	22.6	9,400	--		
Powder platform	24-67	Deutz F6L714 130	52	15,000	17.9	1.4	0.83
Wagner utility vehicle or explosive truck	24-53	Deutz F6L514 70	33	7,270	--0.6	0.50	
Welder-utility truck	24-125	Mercedes-Benz 35	18.3	7,800	--	1.0	0.33
Caterpillar #120	24-D22	Caterpillar D-333A 115	57	14,100	--	3	0.17

(1) Using 6.9 lb/gallon fuel (40° A.P.I. specific gravity - 0.8251)

Condensed and extrapolated from Sutton, G.W., "Industrial Hygiene Survey of U.S. Mines", Denver Tech. Support 1972(21).

(Source: Reference 43)

It was calculated that about 37% of all mine diesel-related emissions were related to the scoop trams, and the scoop tram operator was the person most exposed to diesel pollutants. Scoop trams worked the hardest, picking up ore and operating in dead-end headings where ventilation was poorest, and thus the findings mentioned above are not surprising. The ore haulage cars, although emitting about 37% of the diesel contaminants, operated in well-ventilated haulage ways and cross-cuts and thus their drivers were not exposed to as high a diesel emission level as the scoop tram operators.

A summary of the average CO_2, CO and NO levels recorded are shown in Table 71, together with a calculated value for the concentration which may have been exceeded 1% and 5% of the time.

The average NO_2 concentration was 0.725 ppm, but the level (4.75 ppm) which may have been exceeded 1% of the time was quite close to the TLV. Therefore, this toxicant emerged as the one upon which ventilation requirements should be determined.

Particulate matter concentrations for White Pine, as well as in a second mine, Brushy Creek, are given in Table 72. In both operations, the particulate averaged 1 - 1.5 mg/m^3 and contained about 60% carbon plus hydrogen.

Particulate concentrations were measured at three locations at the same time and included a cross-cut, a drift and a scoop tram. Cross-cut particulate concentrations varied from 0.5 to 1.5 mg/m^3, with an average of 1 mg/m^3. In the drift, it varied from 0.5 to 7 with an average of 3.1 mg/m^3. The LHD operator was exposed to concentrations varying between the two, but were more similar to the values at the drift, being 1 to 6 mg/m^3, with an average concentration of 2.5 mg/m^3.

Additional information on the environmental quality of these mines is given in Section 3.9.5, where the relationship between the various diesel exhaust constituents is discussed.

TABLE 71: Frequency Distributions for Grab Sample Type Measurements at White Pine Mine

Parameter measured	Arithmetic mean	Standard deviation	Geometric mean	Standard geometric deviation	Conc. exceeded	
					5% of time	1% of time
% CO_2 - All	0.128	0.072	0.113	1.64	0.254	0.357
% CO_2 - Headings	0.143	0.089	0.122	1.73	0.300	0.436
% CO_2 - X-cuts	0.114	0.048	0.104	1.54	0.212	0.284
CO, ppm - All	3.92	3.00	3.05	2.20	11.2	19.1
CO, ppm - Headings	4.09	3.23	3.31	2.06	10.9	17.8
CO, ppm - X-cuts	3.76	2.78	2.84	2.31	11.3	19.9
NO, ppm - All	4.82	3.72	4.08	2.01	12.9	20.7
NO, ppm - Headings	5.31	4.28	4.38	2.08	14.6	24.0
NO, ppm - X-cuts	4.35	3.06	3.82	1.92	11.2	17.4
CO + NO, fraction of TLV* value						
- All	0.335	0.162	0.298	1.65	0.68	0.95
- Headings	0.370	0.190	0.326	1.67	0.75	1.067
- X-cuts	0.299	0.128	0.270	1.64	0.60	0.84

(*) TLV is 50 ppm for CO, 25 ppm for NO, but is calculated for an 8-hour time-weighted average measurement while the above data are short-term grab samples.

(Source: Reference 44)

TABLE 72

Particulate Matter Concentrations and Percentage of C + H for Various Locations at the White Pine and Brushy Creek Mines

White Pine Mine

Location of sampling	No. of samples	Conc. of Total particulate matter in air, mg/m^3			Percent carbon + hydrocarbon in particulate matter		
		Avg.	Min.	Max.	Avg.	Min.	Max.
Stamler	4	1.4	0.24	3.32	35	18	61
On tram	3	1.19	0.86	1.44	66	58	72
Near mucking	9	1.18	0.51	1.83	61	32	93
Near Jumbo	6	1.26	0.72	2.24	59	38	99
Total for mine	22	1.23			57		

Brushy Creek Mine

Location of sampling	No. of samples	Avg.	Min.	Max.	Avg.	Min.	Max.
Bottom level exhaust air	9	0.82	0.56	1.0	51	41	66
On loader	5	2.16	1.05	3.68	53	27	75
On jumbo	6	1.57	0.95	2.61	74	65	84
At dump	4	2.28*	1.13	4.65	50	40	56
Near mucking	18	1.53	0.92	2.08	69	58	90
Total for mine	42	1.53			62		

(*) 1.50 mg/m^3 without max value of 4.65

(Source: Reference 44)

Mogan and co-workers (454) recently published additional PAH levels for a Sudbury hard rock mine, an Elliot Lake uranium mine and a salt mine. Table 73 gives total PAH and BaP concentrations in the ambient air of the 3 dieselized underground mines (only include the first 3 lines, just the PAH, carcinogens and benzo(a)pyrenes - not the ratios). Comparisons of these mine atmosphere PAH levels with ambient levels in several Canadian cities will be discussed in Section 4.6. Review of the numbers shown in Table 73 for the hard rock mine suggest that there was a substantial reduction in total PAH content between the winter of 1979/80 and that of 1980/81 with levels in 1980/81 being roughly 15% of those in 1979/80. This reduction in the non-carcinogenic PAH's was paralleled by a reduction in the four carcinogenic PAH's which were measured as well as the BaP levels. No suggestion as to the reason for the differences in the PAH levels during the two timeframes were given.

Recently, Reinbold, Carlson and Johnson (45) re-discussed the results of their 1975 - 1976 surveys of the White Pine Copper and the Brushy Creek lead mines. In their more recent publication the data is expressed in alternate ways with ranking using three criteria to estimate which contaminants are potentially the most critical to control.

The pollutant concentrations are given in Table 74 and the order of ranking appears in Table 75.

In all three instances (1976 Brushy Creek Mine, 1975 White Pine Mine and 1976 White Pine Mine), CO_2 and particulate matter were the most critical pollutants.

The percent increase in the pollutant concentration from the 1976 to the 1975 White Pine Mine data compares "typical" to "worst" data, respectively. Ranking from worst to best was CO, particulate matter, NO and CO_2. Thus, as underground conditions become worse and pollutant levels increase, CO and particulate matter are the critical pollutants, with CO_2 being of the least concern.

Table 73: Total PAH's, Carcinogenic PAH's, and Benzo(a)pyrene Concentrations in the Ambient Air of a Number of Dieselized Underground Mines, ng/1000 m^3, Plus a Comparison with Winter Levels in Hamilton and Sarnia

Site		Hardrock mine 1979-80	Hardrock mine 1979-80	Hardrock mine 1979-80	Uranium mine 1979-80	Uranium mine 1979-80	Salt mine I 1979-80	Salt mine I 1979-80	Salt mine I 1979-80	Salt mine I 1979-80	Salt mine I 1979-80	Hardrock mine 1980-81	Hardrock mine 1980-81	Hardrock mine 1980-81
	ΣPAH's (7)	41388	66551	24670	36186	28501	12379	5538	21883	12369	13854	7528	6260	6982
	Σcarcinogens(4)	30814	56549	20031	31333	22213	9639	4213	18940	6410	12628	6295	4991	4879
	benzo(a)pyrene	8368	4603	3235	1893	3963	539	276	156	382	194	433	1602	1566
Ratio to average winter exposure, Hamilton, Ontario 1975-76	ΣPAH's (7)	2.8	4.4	1.6	2.4	1.9	0.8	0.4	1.5	0.8	0.9	0.5	0.4	0.5
	Σcarcinogens(4)	2.6	4.9	1.7	2.7	1.9	0.8	0.4	1.6	0.5	1.1	0.5	0.4	0.4
	benzo(a)pyrene	3.1	1.7	1.2	0.7	1.5	0.2	0.1	0.1	0.1	0.1	0.2	0.6	0.6
Ratio to average winter exposure, Sarnia, Ontario 1975-76	ΣPAH's (7)	13.8	22.2	8.2	12.0	9.5	4.1	7.3	6.3	2.1	4.2	2.5	2.1	2.3
	Σcarcinogens(4)	11.6	21.3	7.5	11.8	8.4	3.6	1.6	7.1	2.4	4.8	2.3	1.9	1.8
	benzo(a)pyrene	21.3	11.7	8.2	4.8	10.1	1.4	0.7	0.4	0.9	0.5	1.1	4.1	4.0
	ΣPAH's (7)	3933	20760	18300	58380	9360	7950	13630	9250		9780	4000	20760	28550
	Σcarcinogens(4)	2961	18560	12450	52300	6120	5850	10460	7670		7460	2310	18560	20440
	benzo(a)pyrene	546	1410	1080	2300	1670	1040	2210	980		1040	750	1410	3890
Ratio to average winter exposure, Hamilton, Ontario 1975-76	ΣPAH's (7)	0.3	1.4	1.2	3.9	0.6	0.5	0.9	0.6		0.6	0.3	1.4	1.9
	Σcarcinogens(4)	0.3	1.6	1.1	4.5	0.5	0.5	0.9	0.6		0.6	0.2	1.6	1.8
	benzo(a)pyrene	0.2	0.5	0.4	0.8	0.6	0.4	0.8	0.4		0.4	0.3	0.5	1.4
Ratio to average winter exposure, Sarnia, Ontario 1975-76	ΣPAH's (7)	1.3	6.9	6.1	17.4	3.1	2.6	4.5	3.1		3.2	1.3	6.9	9.5
	Σcarcinogens(4)	1.1	7.0	4.7	19.7	2.3	2.2	3.9	2.9		2.8	2.4	7.0	7.1
	benzo(a)pyrene	1.4	3.6	2.7	5.8	4.2	2.6	5.6	2.5		2.6	1.9	3.6	9.9

(Source: Reference 454)

TABLE 74

Concentrations Used to Rank Pollutants at the White Pine and Brushy Creek Mines

	CO_2		CO		NO		NO_2		PM	
Data Group	% by vol.	% of TLV*	ppm	% of TLV*	ppm	% of TLV*	ppm	% of TLV*	mg/m^3	% of TLV**
Brushy Creek 1976	0.15	30	8.5	17	4.8	20	0.73	15	1.53	30
White Pine 1976	0.15	30	4.5	9	5.7	23	--	--	1.23	25
White Pine 1975	0.18	36	8.9	18	8.6	34	--	--	2.23	45
% increase from WPM 1976 to WPM 1975		20		100		48		--		80

* TLV is 50 ppm for CO, 25 ppm for NO, 5 ppm for NO_2 ("C" Limit), and 0.5% by volume for CO_2. Monitoring data are normally based on an 8-hour time weighted average measurement while these data are for short term grab samples. The TLV is basically being used in this context to normalize the data.

** TLV of 5.0 mg/m^3 used for total respirable particulate matter.

(Source: Reference 45)

TABLE 75

Pollutant Ranking from Highest Percent of TLV Level* to Lowest Percent of TLV Level, Based on Short-Term Grab Sample Data Collected in the White Pine and Brushy Creek Mines

	RANKING CRITERIA				
Pollutant Ranking Scale	Averages of BCM 1976 data	Averages of WPM 1976 data***	Averages of WPM 1975 data***	Percent increase from WPM 1976 to 1975 data**	Percent Increase from WPM 1976 to 1975 Data Using a TLV of 2.0 mg/m^3 for PM, and 1.0 ppm for NO_2**
Most Critical Pollutant (Highest % of TLV)	CO_2 - PM	CO_2	PM	CO	CO
	NO	PM	CO_2	PM	PM
	CO	NO	NO	NO	NO_2
Least Critical Pollutant (Lowest % of TLV)	NO_2		CO	CO_2	NO

* TLV is 50 ppm for CO, 25 ppm for NO, 5 ppm for NO_2 ("C" limit), and 0.5% by volume for CO_2. Monitoring data are normally based on an 8-hour time weighted average measurement while these data are for short term grab samples. The TLV is basically being used in this context to normalize the data.

** Brushy Creek Mine NO_2 data used.

*** NO_2 not ranked because it wasn't measured at WPM.

(Source: Reference 45)

The third criteria used the 1 ppm ceiling for NO_2 and a 2 mg/m^3 TLV for particulate matter. In this case the ranking became CO, particulate matter, NO_2 and CO_2.

It was considered that in both mines the pollutants which would be most critical to control would be NO_2 and particulate matter.

It was also demonstrated that a health hazard was most likely to occur in an area which was difficult to ventilate, such as a dead end heading. The authors suggested that an average pollutant concentration value would not necessarily identify the hazard if it was due to an excursion over a ceiling limit (i.e. NO_2). A frequency distribution was considered useful in identifying problem areas and they recommended the use of the estimated 1% frequency concentration (i.e. the concentration exceeded 1% of the time) based on lognormal distribution plots of mine air pollutant values (45).

As examples, the authors provided the following summary tables (Tables 76, 77 and 78):

TABLE 76

Arithmetic Average Concentrations, % of TLV Level

	CO_2	CO	NO	NO_2
WPM, 1975	36	18	34	--
WPM, 1976	30	9	23	--
BCM, Spring	34	19	22	19
BCM, Summer	26	16	12	9

(Source: Reference 45)

TABLE 77

Maximum Measured Concentration, % of TLV Level

	CO_2	CO	NO	NO_2
WPM, 1975	118	140	128	--
WPM, 1976	108	32	72	--
BCM, Spring 1976	68	52	40	70
BCM, Summer 1976	64	40	54	50

(Source: Reference 45)

TABLE 78

Concentration Exceeded 1% of Time, % of TLV Level

	CO_2	CO	NO	NO_2
WPM, 1975	138	214	218	--
WPM, 1976	102	42	98	--
BCM, Spring	70	87	60	131
BCM, Summer	74	63	96	54

(Source: Reference 6)

Schock, Johnson and Bunting (166) described the results of monitoring diesel emissions in a dead-ended drift of a mined out portion of the White Pine Copper Mine in order to determine the relative merits of ventilation configurations, including no auxillary ventilation, induction ventilation at the entrance to the drift, and ventilation of the dead-ended drift with positive displacement tubing. The authors' were mainly concerned with two cases in the load-haul-dump cycle which included:

1. vehicle operator exposure in a heading while mucking, and
2. vehicle operator exposure in a heading while moving down a haulage way in the same direction as the air flow.

The latter assumed that the velocity of the tram was less than the velocity of the air and that the exhaust was discharged upwind of the operator. These two situations are depicted in Figure 81 as Case 1 and Case 2, respectively. A Wagner scoop tram powered by an F8L-714 Deutz air-cooled diesel engine was used. The vehicle was operated in the normal tramming cycle and served as the transient moving source to be modelled later (167). The vehicle speed, operating modes, engine speeds, rack positions, etc., were all monitored. A summary of the experimental set-ups used in these studies is given in Table 79 and Table 80 lists the engine and vehicle operating characteristics during tramming in the dead-ended drift. The engine operated with an average "percent of full load" as follows; 85% while the bucket was being loaded, 63% while the loaded tram

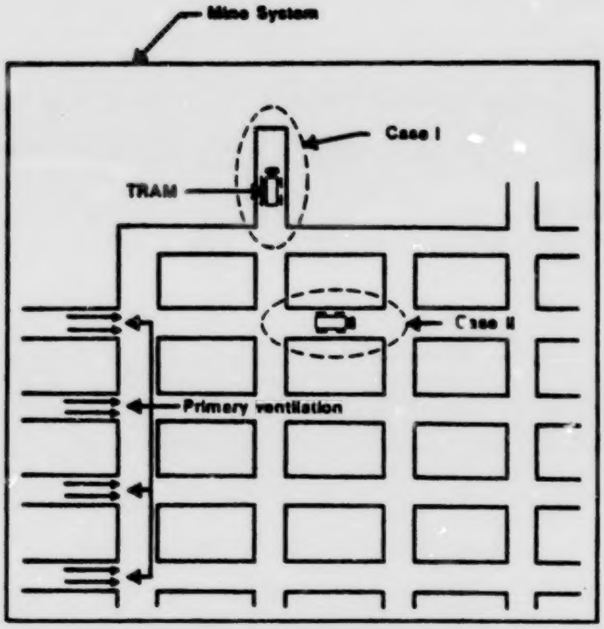


FIGURE 81

Schematic of Mine System

(Source: Reference 166)

was moving out of the heading, and 46% while the vehicle was moving empty into the heading. These three operations represent duty cycle modes 2, 3 and 1, respectively and are typical of LHD cycling used at the White Pine operation.

It was found that induction ventilation (a fan placed on the upwind side of the drift at the entrance to the heading and on the floor) in a down-sloping drift caused higher levels of pollutant concentration than when no auxillary ventilation was used. This was thought to result from recirculation by the induction fans of a fraction of the contaminated heading air. The comparison was made between area and personal monitoring of pollutants. For ventilation configurations where appreciable concentration gradients existed along the length of the drift, the use of area monitors to predict personal exposure was not adequate. With auxillary ventilation or

TABLE 79: Summary of Experimental Work

Experiment Number	Date	Type Of Vent	Average Fuel Flow Gal/hr (1/hr)	Average Time In Heading (Sec/ Cycle)	Average Time Out Heading (Sec/ Cycle)	Total Fuel Consump-Tion in Heading Gal (liters)	Started Mucking At Feet Into Drift (meters)	Stopped Mucking At Feet Into Drift (meters)	Mucking Time (Min)
2001	7-06-78	N.A.V.**	8.42 (31.87)	71	134	2.51 (9.50)	105 (32)	135* (41)	59
2002	7-11-78	N.A.V.	8.58 (32.48)	72	130	2.58 (9.77)	105 (32)	145* (44)	88
2006	8-03-78	Vent Tubing Used	7.94 (30.06)	69	128	2.26 (8.56)	105 (32)	130 (40)	55
2007	8-03-78	N.A.V.	7.57 (28.66)	74	127	2.36 (8.93)	130 (40)	145 (44)	50
2008	8-08-78	Induction Vent Fan	7.20 (27.25)	65	134	1.98 (7.50)	110 (34)	135 (41)	70
2009	8-10-78	Vent Tubing Used	7.48 (28.31)	71	138	2.19 (8.29)	110 (34)	125 (38)	60

* Estimated

** No Auxiliary Ventilation - N.A.V.

(Source: Reference 166)

TABLE 80: Vehicle Operating Parameters

Means/Standard Deviations for 15 Cycles (Average)

	Fuel Flow lbm/min (kg/min)	Engine Speed RPM	BHP HP (KW)	Full Load (%)	Vehicle Velocity MPH (m/s)	Time in Drift (sec)	Time out of Drift (sec)
Mode 1	0.76/0.23 (0.34/0.10)	2213/274	77.0/28.0 (57.4/20.9)	45.7/19.2	2.32/0.53 (1.04/0.24)	29.24/2.88	130.60/11.28
Mode 2	0.97/0.31 (0.44/0.14)	1755/243	122.0/37.0 (91.0/27.6)	85.2/22.2	--	14.16/7.60	
Mode 3	1.02/0.15 (0.46/0.07)	2268/210	108.0/24.0 (80.5/17.9)	62.6/15.9	2.37/0.38 (1.06/0.17)	28.91/2.47	

(Source: Reference 166)

induction ventilation area monitor results were within 11% and 0% of personal monitored results, respectively. The gas monitored was CO_2, and as fuel flow could only be predicted to within 11%, a discrepancy of less than 11% was adequate. Fuel burned at the face was a very important parameter in terms of operator exposure, as exhaust gasses generated at the face had to be transported the greatest distance to be moved out of the heading. The dose and volume of exhaust was greater when the exhaust pipe was out the aft end of the tram rather than at the side of the tram. Thus pollutant levels experienced by the operator will be lower when the exhaust pipe is placed out the rear of the vehicle. It was found that ventilation with positive displacement tubing provided the best removal of diesel-generated pollutants. Induction ventilation appeared to be the least effective of the three methods examined. The comparison between personal and area monitoring showed that in regions of high concentration gradients it was not possible to get good agreement between the two methods, whereas in a well mixed area, good agreement was obtained.

Shock and Johnson (167), using the experimental values derived from earlier work (166), applied computer-derived mathematical modelling to determine the effect of different types of ventilation in affecting diesel pollutant levels in a dead-ended drift. The model predictions were compared to experimental measurements. The cases of no auxillary ventilation and ventilation with tubing were mathematically modelled, using a turbulent dispersion scheme. The scoop tram was modelled as a variable speed, variable strength source of pollutants moving in and out of the dead end drift.

It was concluded that when modelling a load-haul-dump cycle it is necessary to use a model that has the capability of predicting changes in source strength, as well as source location, of pollutants. It was also determined that four relevant factors included effective heading height, apparent longitudinal diffusion coefficient, transverse concentration gradient at the face, and the average dispersion width of the exhaust jet. It was calculated, using a calibrated model based on CO_2 measurements, that it was possible to predict pollutant concentrations at any location in the heading using an engine map and a previously calculated flow field. It was shown that the exhaust pipe location could have a significant impact on operator exposure to exhaust gasses. It was also shown that NO concentrations varied directly with CO_2 levels. Models such as the one described in the paper are useful in assessing the effects of changes in exhaust system configurations and variations in tramming schemes. It was demonstrated that ventilation with tubing resulted in a 50% pollutant exposure reduction, compared to auxillary ventilation.

Using three different tramming schemes the trade-off between productivity and operator CO_2 exposure could be evaluated. The model of loading the ore car at the drift showed the highest CO_2 concentration, and the highest productivity. The use of ventilation tubing reduced the overall concentration by 50 -60% over the no auxillary ventilation case for all three mining schemes. It was found that gas stratification plays an important role in the operator's exposure while he is working a heading with diesel-powered equipment. When cooler air is brought into the face area by ventilation tubing a layering effect tends to diminish the effectiveness of the ventilation system.

Hews and Rutherford (51) provided some details on environmental contaminants at the INCO Sudbury mines. As of 1973, about 5.5×10^6 cfm of fresh air was being supplied to INCO mines. This represented about 80 cfm per ton of ore hoisted per day. Data from weekly gas tests carried out during the latter part of 1972 are given in Table 81.

Most of the ventilation volumes measured at the diesels were of the order of 100 cfm/bhp.

In 1972, gravimetric dust sampling of the breathing zone of the LHD operators was carried out. The findings are listed in Table 82, and resulted in the conclusion that the respirable portion of the carbon particles in the air from LHD exhausts was in the range of 1 - 1.5 mg/m^3.

At the 1977 - NIOSH Diesel Workshop the environmental characteristics work group (18 members), many who are knowledgeable and experienced in analytical methods for pollutants, and in mine atmosphere contaminant levels, provided the data shown in Table 83.

TABLE 81

Diesel Exhaust Contaminants at INCO Mines

1. Adjacent to LHD Operator	95% of Values	99% of Values
CO	trace - 5 ppm	10 ppm
NO_2	trace - 1 ppm	2 ppm
Aldehydes	nil - trace	1 ppm

2. In General Atmosphere

CO	86%: trace to 2 ppm 98%: 5 ppm
CO_2	96%: 400 - 1000 ppm 99%: 1500 ppm

(Source: Reference 51)

TABLE 82

Gravimetric Dust Sampling Data for INCO Mines (1972)

Diameter Filter	Sampling times	Total Respirable Dust (mg/m^3)	Rock Dust (mg/m^3)	Combustible Dust (mg/m^3)
25 mm	3 hours	2.9	1.3	1.6
37 mm	8 hours	2.0	0.5	1.5
37 mm (non-diesel areas)	8 hours	0.9	0.45	0.45

(Source: Reference 51)

TABLE 83: Coal Mine Exposure Concentrations Estimated at 1977 - NIOSH Diesel Workshop

(NOTE: Values in this table have limitations and should be only considered as estimations subject to change as more accurate data become available.)

COMPONENT	RANGE, UNDILUTED (1)	EXPOSURE VALUES Median Value	 Working Range	CONFIDENCE IN EXPOSURE VALUES	CALC. DILUTION FACTOR (AVERAGE)	COMMENTS
CO_2	6.1 - 7.2%	700	400 - 1100 ppm	Good	(165)	Dilution factor is baseline for comparison of all values. Exposure values include 300 ppm background CO_2.
CO	114 - 513 ppm	10	0 - 20 ppm	Good	10 - 50 (30)	Suggest undiluted values low.
NO	225 - 735 ppm	3	1 - 5 ppm	Good	75 - 245 (160)	
NO_2	36 - 84 ppm	0.3	0.1 - 0.5 ppm	Good	120 - 280 (200)	
SO_x	7.7 ppm	—	0.5 ppm	—	—	No mine data.
SO_x (2)	40 ppm	—	0.5 ppm	Good upper limit	80	No mine data. Based on 0.2 sulphur in fuel.
H_2SO_4	—	—	0.2 mg/m^3	Unknown	—	Used 10% of SO_x values as basis.
Hydrocarbons	(3)	—	5 - 20 ppm	Poor	—	Measured values as methane, but univerified (Reference 1233)
Benzene	(3)	—	0.01 ppm	—	—	Rough estimate.
Aromatics	—	0.7	0.13 - 2.1 mg/m^3	Unknown	—	Unpublished Bureau of Mines data.
Oxygenates	(3)	0.04	0.006 - 0.11 mg/m^3	Unknown	—	Unpublished Bureau of Mines data. Does not contain formaldehyde.
Total Aldehydes	(3)	0.045	0.007 - 0.19 mg/m^3	Unknown	—	Unpublished Bureau of Mines data.
Formaldehyde	(3)	0.04	0.007 - 0.06 mg/m^3	Unknown	—	Unpublished Bureau of Mines data.
Diesel Particulate	0.12 - 0.47 g/bhp-hr	—	0.1 - 1 mg/m^3	Unknown	—	
Respirable Coal Particulate	—	1.3	0.1 - 10 mg/m^3	Good	—	Compliance values.

OTHERS: PNA, Organic Acids, Organo-Sulphur Compounds, Organo-Nitrogen Compounds, HNO_3, HNO_2, Trace Metals : Insufficient data to generate exposures.

FOOTNOTES: (1) Emission work group data; (2) Without Scrubber; (3) Emission work data questionable or no data.

The values are based on concentrations, either measured in coal mines or calculated from figures provided by the Emission Work Group at the same meeting. Using these figures, NO_2 is the component upon which it would seem appropriate to set ventilation requirements. The group recommended that, "until reliable emission data becomes available for some of the components, the median CO_2 dilution factor (140) can be used to derive rough estimates of exposure from source (tailpipe) concentrations in the absence of evidence to the contrary".

These values appear, by comparison to the data reported in the various studies discussed above, to be fairly representative of the situation in most mines employing heavy-duty diesel engines. Obviously, they must be modified when actual measurements are available for any particular mine, but they serve as useful and realistic values for general discussions and recommendations relating to the protection of the miners' health.

Significant new information relating to the mining environment comes from Westaway's group at Laurentian University in Sudbury who have measured PAH concentrations in several Ontario mines.

Faulds, Waszczylo and Westaway (164) are, to our knowledge, the first to quantitate the level of PAH's (PNA's) in Canadian mine atmospheres. Following collection on fibreglass filters through high-volume samplers, the particulate matter was extracted with various solvents and the PAH's separated using thin-layer chromatography followed by quantitative analysis by spectrofluorometry. Samples were collected from one of Inco's Sudbury nickel mines and in the Rio Algom uranium mine at Elliot Lake.

It was found that as the work area ventilation increased, the concentration of the PAH's decreased, although some of the individual PAH's departed from this general rule (Table 84). The effects of variable diesel activity at a constant ventilation air flow suggests that a correlation exists between diesel activity and the PAH concentration (Table 85).

Air samples taken in a salt mine showed that the air in the mine contained significantly higher concentrations of PAH's than the incoming air. In fact, the PAH's near the working sites ranged from 21

TABLE 84

Concentrations in ng/1000 m^3

Pyrene	168,107	100,394	22,388
Fluoranthene	72,747	20,637	8,337
Benzo(a)anthracene*	9,760	4,489	1,044
Benzo(e)pyrene	26,880	17,462	2,600
Perylene	1,467	635	100
Benzo(k)fluoranthene	3,120	5,228	526
Benzo(a)pyrene*	1,893	3,963	375
Benzo(ghi)perylene*	15,387	10,943	1,482
Dibenzo(def,mno)chrysene	266	425	48
Naptho(1234,def)chrysene*	12,613	6,640	900
Benzo(rst)pentaphene*	1,440	667	124
Total Concentration of PAH	313,680	171,483	37,924
Total Conc. of Carcinogens	41,098	26,702	3,925
Percent CO_2 (Corrected)	0.30%	0.10%	--
Place Collected	Development heading. Very little Ventilation	Development heading. Ventilation was 45000 cfm to the face. Water was poured on the muck pile.	50 - 55' from the dump. The dump was beside an air raise adding 20,000 cfm of air. Dump was sprayed with water.

(Source: Reference 164)

TABLE 85

Concentrations in ng/1000 m^3

Pyrene	59,826	50,191	6,171
Fluoranthene	--	--	--
Benzo(a)anthracene*	--	--	--
Benzo(e)pyrene	26,593	30,143	10,339
Perylene	1,250	1,127	653
Benzo(k)fluoranthene	7,345	7,489	3,113
Benzo(a)pyrene*	8,368	4,603	3,235
Benzo(ghi)perylene*	16,905	36,324	10,677
Dibenzo(def,mno)chrysene	4,895	12,645	5,413
Naptho(1234,def)chrysene*	4,895	12,645	5,413
Benzo(rst)pentaphene*	646	2,977	706
Total Concentration of PAH	127,807	146,885	41,180
Total Conc. of Carcinogens	30,814	56,549	20,031
Percent CO_2 (corrected)	0.13%	0.09%	--
Diesel Activity	1 ST5 loading, hauling, dumping all shift	Occasional activity by one ST5 at the dump	Intermittent activity far away from the dump

(Source: Reference 164)

- 220 times the concentration of the PAH's in the ventilating air. The "carcinogenic content" was from 21 - 62 (an average of 28) times higher in the mine than outside. It was found that the PAH's were not simply located in or near the actual working area, but were travelling on particulate matter over considerable distances through the mine.

The specific PAH's measured are tabulated in Table 86. It was calculated that the potentially carcinogenic material represented 10 ± 1% of the total PAH's in the samples from the mines where GM-Detroit or Caterpillar diesels were used, whereas in mines where Deutz engines were in use, the carcinogens represented an average of 34 ± 13% of the total PAH's. These data strongly suggest that PAH levels are influenced by different fuels and/or the type of diesel engine.

TABLE 86

Concentrations in ng/1000 m^3

	Intake Air Sudbury	Intake Air Salt Mine	Average Underground Sudbury	Average Underground Elliott Lake	Average Underground Salt Mine
Pyrene	--	9,154	38,729	134,250	280,200
Fluoranthene	--	228	--	46,692	13,521
Benzo(a)anthracene*	--	72	--	7,125	22,402
Benzo(e)pyrene	45	582	22,358	22,171	32,135
Perylene	17	6	1,010	1,051	192
Benzo(k)fluoranthene	74	112	5,982	4,174	6,806
Benzo(a)pyrene*	111	12	5,402	2,928	576
Benzo(ghi)perylene	1,104	106	21,302	13,165	8,955
Dibenzo(def,mno)-chrysene	9	4	1,413	346	532
Naptho(1234,def)-chrysene	73	582	7,651	9,627	8,978
Benzo(rst)pentaphene*	10	82	1,443	1,054	2,658
Total Concentration	1,443	10,994	105,290	242,583	375,078
Total Concentration of Carcinogens	1,298	908	35,798	26,774	43,496
Total PAH excluding Pyrene	1,443	1,840	66,561	108,333	94,878

(Source: Reference 164)

It was noted that in a hard rock mine the PAH's did not seem to move through the same distances as in the salt mine. It was suggested that the differing results may have been related to differences in humidity between the two mines with rapid settling in the hard rock mine as a result of diesel particulate being adsorbed onto mine dust, thus forming heavier particles which settled onto the floor of the drift. The total concentration of carcinogenic PAH's was only 39% higher than the average found in the exhaust when PTX catalytic converters were used and there was no blasting present. The data clearly indicated that PTX catalytic converters do not decrease either the total PAH or the total "carcinogenic" PAH's significantly. Further, the data showed that blasting, as well as diesel activity, contribute to the presence of PAH's in the mine environment.

Additional calculations (165) indicated that the concentration of PAH's were significantly higher in dieselized underground mines than in the air above ground. Individual PAH's were between 6 and 349 (an average of 67) times higher in the mine atmosphere than in the average Sudbury air and from 0.6 to 8 (average of 2.7) times higher than the highest concentrations of PAH's reported in the air over Toronto, Hamilton or Sarnia. Also, the carcinogenic compounds isolated in the mines were between 6 and 350 times more concentrated in the mine than they were in the average Sudbury air, and were from 0.5 to 7 times the maximum concentration detected in the air over the four Ontario cities studied. There was insufficient data to establish whether the PAH concentration correlated with the CO_2 concentration in the dieselized underground mines (Table 87).

Of interest was the finding that it took more than 50 hours of extraction with benzene to remove the "available" PAH's from the filters and additional extractions with methanol and benzene to remove all the PAH's. These data showed that diesel particulate can absorb 1000 times more BaP than has been detected in actual samples, which illustrates the PAH's the strong affinity of PAH's for diesel particulate.

TABLE 87

The Concentrations of Carbon Dioxide, the Total PAHs and Benzo(a)pyrene in Different Samples Collected in Dieselized Underground Mines

Sample	Percent CO_2 (corrected)	Total Concentration[1] of PAH in ng/1000 m^3	Concentration of Benzo(a)pyrene in ng/1000 m^3
Rio Algom 1	0.30	231,173	1,893
Inco 1	0.13	127,807	8,368
Rio Algom 2	0.10	146,357	3,963
Inco 2	0.09	146,885	4,603

[1] The concentrations of fluoranthene and benzo(a)anthracene are not included in the total.

(Source: Reference 165)

The PAH's from the mine samples were considerably different from those collected in the air above ground. For example, the underground samples seemed to contain more impurities and were harder to extract. According to Westaway, of even greater importance, was the fact that the underground samples contained many more PAH's.

Westaway concluded that the Detroit diesels were better suited for mining operations because the average concentration of carcinogenic PAH's was lower in the Elliot Lake than in the Inco samples, where Deutz engines were used. His findings implied that the PAH's were burned more completely by the Detroit diesels and other, indirect, evidence supported this conclusion. For instance, the weights of the benzene extracts were significantly lower in the Elliot Lake samples, suggesting that there was less unburned hydrocarbon in the exhaust. However, it was also pointed out that the Detroit diesels at Rio Algom were almost new, and had run less than 700 hours when the sampling was carried out, whereas the Inco diesels were older engines. It was emphasized that only 11 out of the more than 30 PAH's were analyzed quantitatively. Two other carcinogenic PAH's (benzo(b)fluoranthene and 3-methylcholanthrene), were identified qualitatively. Thus, neither the total number of carcinogens nor the total concentration are known at the present time.

3.9.5 Atmospheric Pollutant Relationships

Rather than having to measure the concentration of all of the toxic constituents of diesel exhaust, there would be obvious advantages if the measurement of one pollutant could be used to predict the levels of all other emission components.

Hurn (53) expressed the view that the measurement of CO_2 would be suitable for such purposes. Carbon dioxide output is directly related to fuel consumption and is an irreducible component of diesel exhaust. The minimum ventilation required to maintain or reduce CO_2 to its acceptable level should result in all other diesel-related pollutants being diluted to acceptable concentrations. Additionally, CO_2 levels are present at an order of magnitude more than other diesel gases, such as CO, and relatively inexpensive, accurate continuous monitoring instruments are available which make CO_2 a particularly attractive component for use as an index of diesel pollution.

Johnson's group from Michigan Technological University examined this concept at White the Pine copper and Brushy Creek metal mines (43, 44). Deutz-powered scoop trams and haulage vehicles comprised about 75% of the diesel power at White Pine and, therefore, the data appear comparable to the Canadian hard rock mine situation.

It was emphasized that "while no perfect linear relationship would be expected to exist between CO_2 and the other contaminants, high levels of CO_2 would definitely warn of potentially high levels of the other contaminants, and particularly of inadequate ventilation" (44).

Simple linear regression analyses were calculated for each airborne contaminant and CO_2. Details can be found in reference (44).

The conditions between the dependent variables and CO_2 were significant for CO, NO and CO + NO, but not airflow.

Table 88 presents the equations for the least square regression lines relating the contaminants listed to CO_2, as well as the predicted CO_2 concentration. The right hand column is of the most interst. The value—predicts the CO_2 concentration at the TLV of the other contaminant at which there is a 95% probability that the contaminant, other than CO_2, is below its TLV.

For example, at a CO_2 level of 1.81%, there is a 95% chance that the CO concentration is below 50 ppm.

Figure 32 shows graphically the relationship between CO_2 and particulate concentrations, and Figure 83, between CO_2 and the combined levels of CO, NO and NO_2.

Also, Figure 84 provides useful data of the particulate exposure in various areas of the mine. The less well-ventilated drifts, where scoop trams operated, consistently had the highest levels. Exposure at the scoop tram was similar, and was related to the particulate at the drift.

The data clearly indicated that for the White Pine Mine, it was more difficult to ventilate for NO than for CO. In order to have a 95% chance that the NO did not exceed its TLV of 25 ppm, the CO_2 would have to be kept below 0.43%, on a time-weighted average basis.

Although more experimental data is required from different types of mines, the available data provide strong support for the concept of using CO_2 and/or CO as an index of other diesel-related pollutant levels.

Following their original work, Johnson has continued to explore the concept of using CO_2 as a "marker" pollutant (9).

The rate of increase in CO_2 concentration is determined by the rate of fuel consumption, the ventilation rate and the volume of air space into which the pollutants are emitted. Thus the CO_2 concentration might be expected to be proportional to the concentrations of other exhaust contaminants, but the others (NO, NO_2 and PM) are affected by engine load and speed.

Based on the White Pine and Brushy Creek Mine data it was calculated that the best relationship was between NO and diesel

TABLE 88: Regression Equations Relating CO, NO, NO_2 and Combined CO + NO Concentrations to CO_2 Along with Prediced CO_2 Concentrations at TLV for These Contaminants

Contaminant of interest	Where CO_2 measured	Equation of regression line	TLV level	CO_2, % On regression line at TLV for contaminant of interest	Co_2, % At which there is a 95% probability that contaminant of interest is at or below TLV
WHITE PINE MINE					
CO, ppm	Crosscuts	CO, ppm = 24.59 (% CO_2) + 0.94	50	2.00	1.81
	Headings	14.97 (% CO_2) + 1.91	50	3.21	2.85
	All	16.81 (% CO_2) + 1.74	50	2.87	2.58
NO, ppm	Crosscuts	NO, ppm = 55.82 (% CO_2) - 2.01	25	0.48	0.43
	Headings	39.55 (% CO_2) - 0.33	25	0.64	0.53
	All	42.93 (% CO_2) - 0.68	25	0.60	0.51
CO + NO (fract. of TLV)	Crosscuts	CO + NO, fract. of TLV = 1.93 (% CO_2) + 0.056	1	0.49	0.40
	Headings	1.52 (% CO_2) + 0.120	1	0.58	0.42
	All	1.67 (% CO_2) + 0.095	1	0.54	0.43
BRUSHY CREEK MINE					
CO, ppm	All	CO, ppm = 55.05 (% CO_2) + 0.178	50	0.91	0.83
NO, Ppm	All	NO, ppm = 37.92 (% CO_2) - 0.058	25	0.67	0.64
CO + NO (fract. of TLV)	All	CO + NO, fract. of TLV = 2.52 (% CO_2) + 0.01	1	0.39	0.32
NO_2, ppm	All	NO_2, ppm = 9.06 (% CO_2) = 0.71	5	0.63	0.58

(Source: Reference 44)

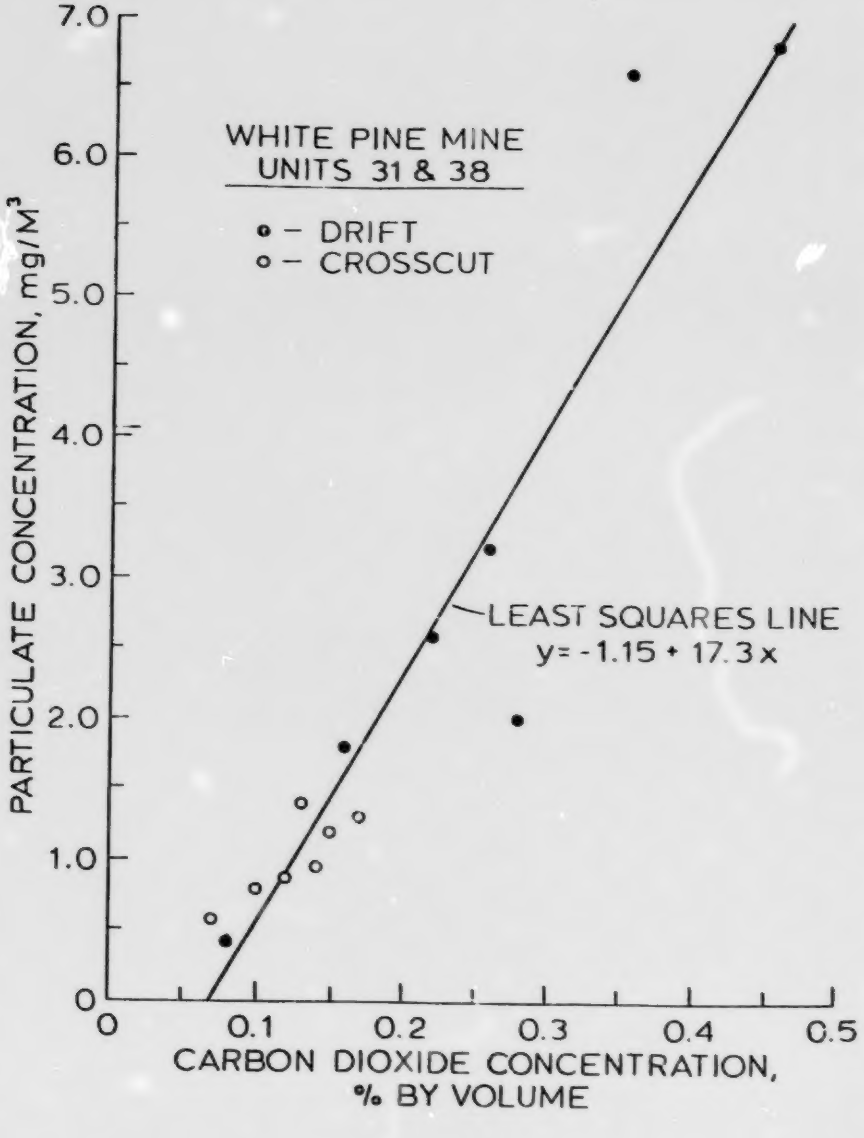


FIGURE 82

Concentration of respirable particulate matter versus average concentration of carbon dioxide for simultaneous measurements at White Pine Mine

(Source: Reference 44)

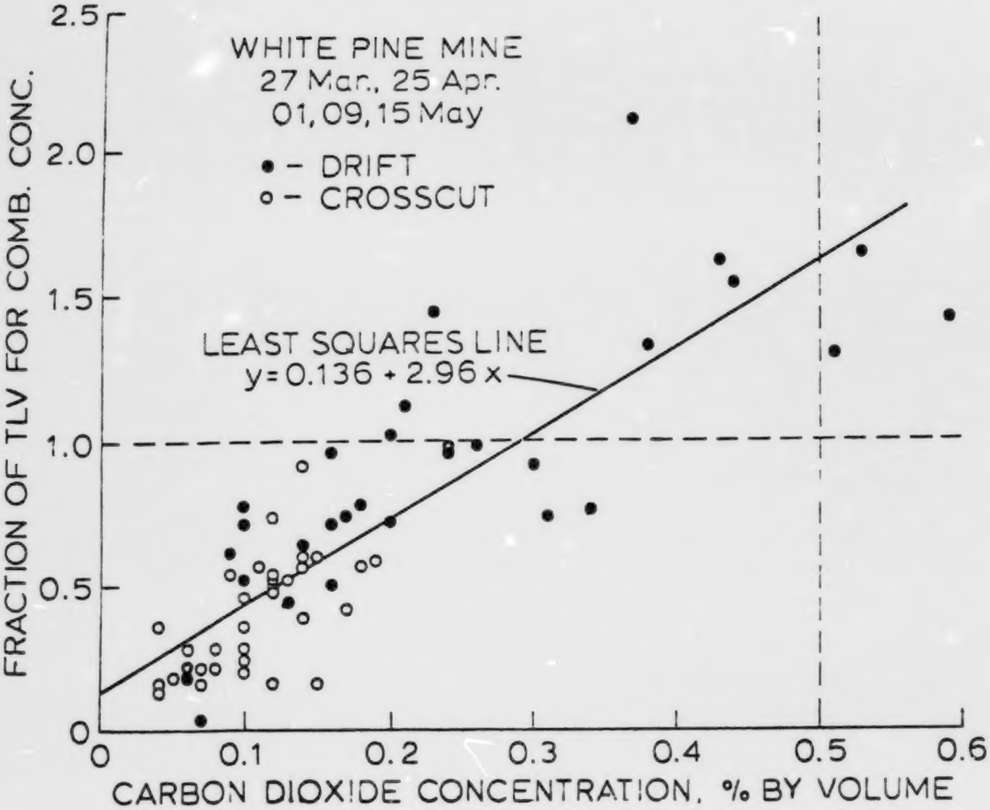


FIGURE 83

Fraction of TLV for combined concentration of CO, NO and NO_2 versus carbon dioxide concentration

(Source: Reference 44)

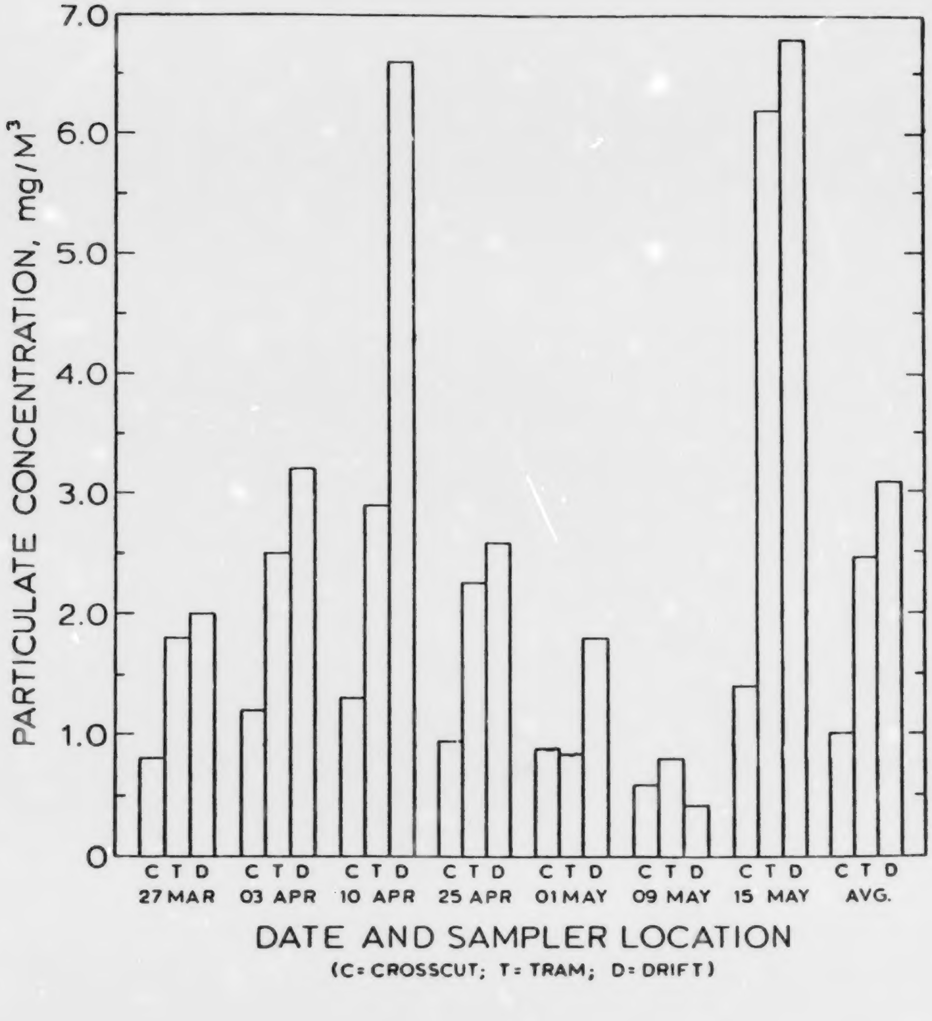


FIGURE 84

Concentrations of particulate matter at cross-cuts, on trams and in drifts

(Source: Reference 44)

particulate matter. There was a fair amount of scatter between CO_2 and CO, which may have been a result of CO released during blasting and not diesel related.

The authors concluded that an accurate estimate of the concentration of other pollutants from CO_2 was not feasible, but CO_2 levels could be selected such that maintenance of levels below a certain concentration would limit the probability that the TLV's of the other pollutants would be exceeded.

3.9.6 Dust or Particulate Matter

Dust is an ever-present and undesirable component of the underground mining environment, with coal dust being associated with black lung disease or pneumoconiosis and quartz with silicosis.

In the dieselized mine, the presence of dust, or more broadly, particulate matter, takes on additional concern because of the added presence of diesel particulate or soot. The characteristics of this particulate was discussed in Section 3.4.7, and will not be reiterated here except to remind the reader of two points. One is that most diesel particulate is respirable, and secondly, it may be similar to activated charcoal in that many diesel contaminants, including toxic gases such as NO_2 and SO_2, as well as acids and carcinogenic substances, such as PNA's, adsorb onto it and are thereby carried into the lungs. In the lungs, the adsorbed substances may be desorbed, or eluted off of the particulate matter, allowing their toxic properties to become apparent. The full significance of the potential for diesel soot to act as a transport media for toxicant delivery to the pulmonary system is not known.

TLV's have been established for silica (see reference 5 for details), with the permitted levels varying with the quartz content of the dust. Respirable coal dust has a TLV of 2 mg/m^3. Nuisance dusts, such as potash, cement, starch, etc., are considered inert and a TLV of 10 mg/m^3 for total nuisance dust or 5 mg/m^3 for respirable nuisance dust, or 30 million or 15 million particles per cubic foot (mppcf), respectively, has been recommended by the ACGIH.

Soot or carbon black has been assigned a TLV of 3.5 mg/m^3. However, whether this limit is applicable to diesel soot is doubtful. Stokinger (171), commented that "the TLV of 3.5 mg/m^3 probably has little relevance to diesel carbon smoke; the value is set for pure carbon particles for aesthetic reasons, and hence does not apply to carbon particles containing adsorbed, potentially toxic substances. The TLV that is ultimately set for diesel smoke must be based on the toxic potential of the combined substances with the carbon".

The TLV for mine air particulate matter is determined by its silica (SiO_2) content. For respirable particles containing greater than 1% SiO_2, the TLV is:

$$\text{TLV} = \frac{10\ \text{mg/m}^3}{\%\ SiO_2 + 2.0}$$

In Canada for respirable particulate matter containing less than 1% SiO_2, the TLV is 5.0 mg/m^3. In the U.S.A., this value is 10 mg/m^3.

The ACGIH has announced its intention to lower the respirable amorphous silica dust TLV to 3 mg/m^3.

As was noted earlier, the sources of particulate matter are primarily ore dust and diesel particulate emissions or soot. As well, oil mist may contribute to the total particulate levels.

No TLV has been set for diesel particulate matter which is considered a part of the nuisance particulate.

Johnson's group (9, 45) attempted to differentiate diesel particulate from other particulate matter (see section 3.3.3) by analysis of C, H and N and by using the C + H content to estimate the percentage diesel related particulate matter. As indicated earlier, a significant carbon content in the mine ore leads to errors in this estimate.

It was found that in the White Pine Copper Mine, the respirable particulate matter samples averaged 0.70 mg/m^3 C + H, and at the Brushy Creek Mine 0.86 mg/m^3 C + H after subtracting 10% for carbon in the dolomite ore or an average of 57 and 62% C + H, respectively.

These same investigators (9, 45) consider the C-H-N analysis of respirable particulate matter a feasible way to estimate the contribution of diesel soot to the total respirable particulate matter. In the two mines examined, the C + H in particulate samples was about 60%, indicating that about 60% of the respirable particulate matter was diesel related. At the face during ore loading, the value was as high as 90% C + H. Thus, the estimated mean diesel particulate values would have been 0.74 and 0.92 mg/m^3 in the general air and 1.1 and 1.35 $mg/^3$ during loading in the White Pine and Brushy Creek Mines, respectively.

A report by MacLaren (136) reviewed the six major sources of airborne dust as summarized below:

Ore Handling: operations such as dumping, loading, mucking, slushing and hauling can generate ore dust which would consist mainly of large solid particles having a composition similar to the ore. Where dust dispersion is suppressed using water sprays, water particles are also added to the mine atmosphere.

Blasting: produces an aerosol composed of pulverized and possibly vaporized ore, plus materials from the blasting component. Because of the large amount of available energy, and possible localized high temperatures, the particle size distribution from this source might be smaller than the normal ore dust.

Drilling: operations can be expected to generate an aerosol composed of ore dust, oil droplets, water droplets and other components. If the oil droplets are assumed to be the only combustible fraction of the dust, it has been suggested that the mass of lubricating oil droplets released into the atmosphere by some drills can exceed the mass of dust particles generated by the drilling operation itself.

Diesel exhaust: contributes small-diameter particles composed mostly of carbon or soot. Liquid particles of fuel or lubricating oil are also emitted, the amount being dependent upon engine operating conditions.

Welding: generates small-diameter particles composed of metals such as manganese, iron and flux components.

Ventilation Air: at the surface, ventilation air contributes all of the components normally present in the local atmosphere with their characteristic distribution, plus any components introduced in heating the incoming air. Underground or in the mill, ventilation air carries with it the original components, plus any particles added by mine or mill processes preceeding the point of observation. Every attempt is made to supply fresh air at all working faces underground or on the surface, but the regulation of flow passages for the air is extremely complex and some contamination of the fresh air usually occurs as the air traverses the mine or mill passages.

The konimeter remains the main instrument for measuring operational dust levels. Air (5 cc) is drawn through a small orifice and impinges on a glass slide coated with an adhesive. The particles stick to the slide which, after heating and treating with acid to remove combustible material (such as diesel soot), and acid solubles (such as carbonates), are counted visually under a microscope or with an electronic scanner.

The total mineral dust concentration is expressed as particles per cubic centimeter (ppcc). The konimeter is considered to measure respirable dust with a size of 0.5 - 5.0 microns (u).

The konimeter uses a "grab" or random spot sample and provides no information on a miner's time-weighted average or work shift exposure, unless frequent random samples of known statistical accuracy are taken.

Alternate dust sampling and measuring devices to the konimeter are available. A review of the available instrumentation can be found in various texts and references, including 21 and 172.

Personal samplers are available which collect the respirable dust on pre-weighed filter paper, silver membrane or glass fibre filters and on which the dust, in mg/m^3, is determined gravimetrically. Some of these methods lend themselves to silica content analyses by x-ray diffraction.

Also, in the case of filters, if the weight of the sample is determined before and after combustion, an estimate of the diesel-derived particulate can be made. It must be remembered, that although diesel soot is combustible, any losses in weight during combustion will include losses of water of crystallization, oil or fuel trapped during sampling, and any combustible ore dust which might be present in the atmosphere. For a dieselized mine, estimates of diesel particulate representing about 80% of the total combustible, respirable dust have been made (121).

Representative konimeter data for the Denison, Elliot Lake uranium mine during 1975 - 1976 are presented in Table 86.

Most dust counts were in the range of 0 - 300 ppcc, but peak levels as high as 434 ppcc have been recorded. A more detailed breakdown of the konimeter data is given in Table 87. The greater number of measurements occurred below 150 and above 300 ppcc. Obviously, there was a wide variability in dust control in the mine. Additional details of the dust levels at the Elliot Lake uranium mines can be found in reference 172.

A note of explanation is in order. High dust levels in Table 83 include pulling chutes, underground crushers and conveyors where use of respiratory protection is required. Also, the 434 ppcc at the shaft loading pocket refers to levels outside the operator's booth, which is equipped with a filtered air supply. Graphs of average dust levels provided by Denison (150) show a decreasing trend since 1976 (Figure 85).

The decreased dust load is related to the increased ventilation installed at the Elliot Lake mine. In the early 1970's, the mines were provided with air at about 0.625×10^6 cfm. By 1978, this had been increased to 2.1×10^6 cfm and in 1983 is about 4.5×10^6 cfm (Figure 85).

It was calculated that in 1978 for every ton of ore mined at Denison 12 tons of air is forced underground, which represents 1400 cfm per man working underground (172).

TABLE 86

Representative Dust Monitoring (Konimeter) Date - Mine[1]

	% of Total Samples**	Percent of Samples Per Range of PPCC*				Average PPCC
		0-300	300-500	500-800	800+	
Headings						
- Drilling	9	79	14	7	0	196
- Loading	6	67	25	7	1	256
Raises						
- Drilling	4	84	8	8	0	179
- Scraping	3	54	34	9	3	301
Stopes						
- Drilling	12	77	15	8	0	192
- Scraping	10	78	17	4	1	162
Dumping	6	81	9	8	2	203
Pulling Chutes	5	79	10	10	1	203
Underground Crushers	7	69	24	5	2	241
Underground Conveyors	6	71	16	11	2	249
Shaft Loading Pockets	3	43	16	29	12	434
Fresh Air Splits	2	100	0	0	0	59
Return Air Splits	2	88	4	2	6	187
Shafts and Stations	7	82	15	3	0	173
Plain Drifts and X-Cuts	18	94	4	2	0	84

* Konimeter slides counted after heat treatment (>1000°F). Combustibles driven off and counts normally assumed to represent silica particles.

** Number of samples taken in six month period typically range between 400-600 per mine.

[1] Data compiled from one year of konimeter surveys at Elliot Lake Mine (1975-1976)

(Source: Reference 150)

TABLE 87

Representative Dust Monitoring (Konimeter) Data - Mill[1]

	% of Total Samples**	Percent of Samples Per Range of PPCC*				Average PPCC
		0-300	300-500	500-800	800+	
Primary Crushing	4	58	26	16	0	233
Secondary Crushing	11	88	11	1	0	202
Screening	9	88	11	1	0	182
Conveying and Transfers	13	66	21	12	1	273
Wet Mills and Concentrators	37	99	1	0	0	102
Assay Laboratories	5	100				97
Shops	21	95	4	1	0	95

* Konimeter slides counted after heat treatment (>1000°F). Combustibles driven off and counts normally assumed to represent silica particles.

** Number of samples taken in six month period typically range between 250-350 per mine.

[1] Data compiled from one year of konimeter surveys at Elliot Lake Mine (1975-1976)

(Source: Reference 150)

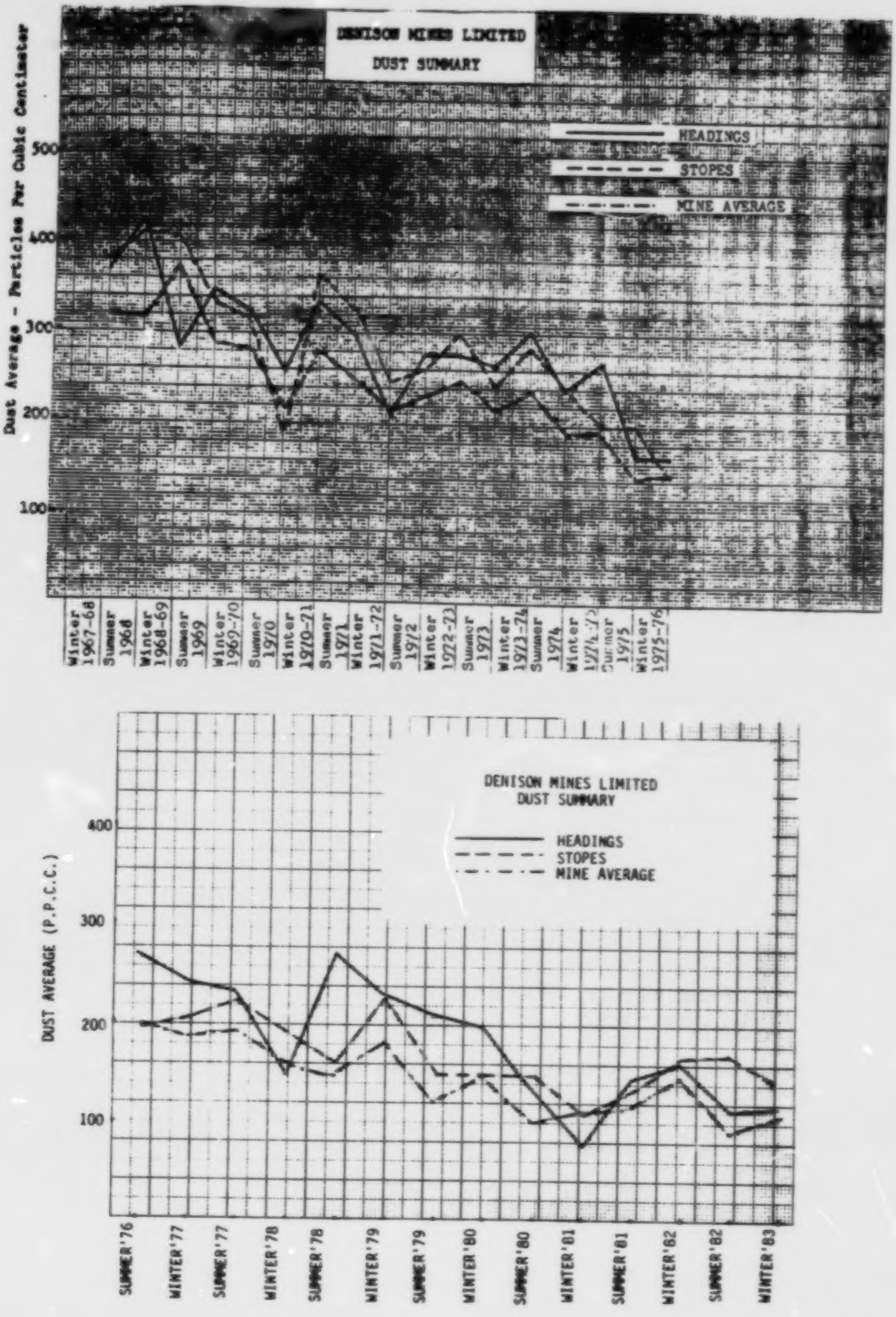

DENISON MINES LIMITED
DUST SUMMARY
HEADINGS
STOPES
MINE AVERAGE
Dust Average - Particles Per Cubic Centimeter
500
400
300
200
100
Winter 1967-68
Summer 1968
Winter 1968-69
Summer 1969
Winter 1969-70
Summer 1970
Winter 1970-71
Summer 1971
Winter 1971-72
Summer 1972
Winter 1972-73
Summer 1973
Winter 1973-74
Summer 1974
Winter 1974-75
Summer 1975
Winter 1975-76
DENISON MINES LIMITED
DUST SUMMARY
HEADINGS
STOPES
MINE AVERAGE
DUST AVERAGE (P.P.C.C.)
400
300
200
100
SUMMER'76
WINTER'77
SUMMER'77
WINTER'78
SUMMER'78
WINTER'79
SUMMER'79
WINTER'80
SUMMER'80
WINTER'81
SUMMER'81
WINTER'82
SUMMER'82
WINTER'83

FIGURE 85

Knight et al (173) measured respirable dust in 6 Ontario mines. Table 88 gives the proportion of quartz and combustibles found in the total respirable samples. What proportion of the combustibles was derived from drill aerosols cannot be estimated as the "work place" was not defined. Graphs of total respirable dust versus respirable quartz (Figure 86) showed a large variation or lack of correlation between the two. This lack of correlation was true for mines with low, medium and high quartz contents. The variation was likely due to diesel exhaust particulate.

Using published values for these same mines, together with numbers for the dust concentrations in non-dieselized sections of the mines, it was concluded that the respirable portion of the carbon particles in the air derived from LHD diesel engines was in the range of 1.0 - 1.5 mg/m^3.

TABLE 88

Composition of the Respirable Dust

	Quartz %			Combustibles %		
		Range			Range	
Site	Mean	Min.	Max.	Mean	Min.	Max.
Mine A, B and C Working place	27.7	0	69			
Mine C only Working place	18.0	0	41	65	11	96
Mine D Working place	1.5	0	2.3			
Mine E Working place	6.5	0	20.6	48.2	5.4	97.5
Mine F Working place	6.4	0	12.8	55.5	36.3	70.4

Approximate amount of quartz in ore is 60-65% in Mines A, B, C, 8% in D and 20% in E and F.

(Source: Reference 173)

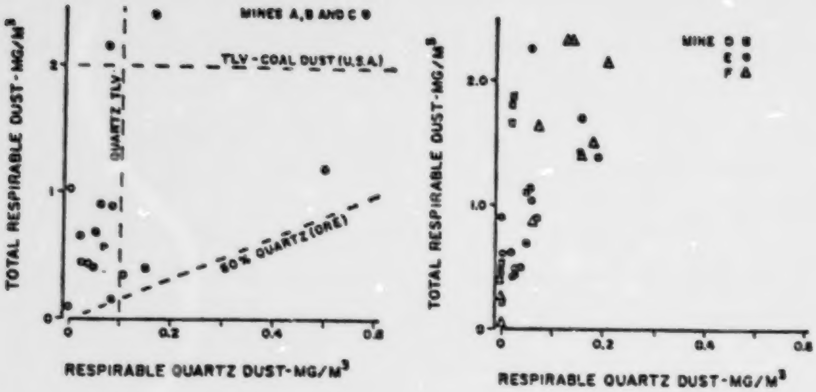


FIGURE 86

Total respirable dust versus respirable quartz

(Source: Reference 173)

Although detailed survey data are available for Denison Mines, and have been presented earlier, any further discussion is not warranted because of the increased volumes of ventilation. In effect, all previous data is irrelevant to the current situation at the Denison Mines site.

The most recent particulate data available for Denison was provided by the Company from personal gravimetric sampling reported in July, 1977. However, the data was not detailed as to site. Quartz dust varied from 0 to 0.82 mg/m^3, depending on the sampling site. The mean quartz percentage of the total respirable dust was estimated as being about 77%. It was calculated that at the 90% confidence level, the dust samples analyzed contained between 28 and 36% quartz.

A summary of the semi-annual underground survey at Denison Mines for the period ending September 30, 1977 can be seen in Table 89. Of the 532 samples analyzed, 79% had counts of 0 - 300 ppcc, 11%

had 300 - 500, 6% 500 - 800 and 3% were over 800 ppcc. In the latter instances, most of the samples were taken at shaft loading pockets, conveyors and crushers, where the operators are either provided with environment-controlled cabs or with personal protection devices (Note: the personal protection devices are provided but their use is not mandatory).

TABLE 89

Summary of Semi-Annual Survey of Air Conditions

(Period Ending September 30, 1977)

FIRM DENISON MINES LIMITED MINE OR PLANT DENISON MINES LIMITED

	OPERATION		NO. ACTIVE WORKINGS SAMPLED	DUST - P.P.C.C.									
				NUMBER OF SAMPLES	AVERAGE	NO. OF SAMPLES				AIR		FILTERED AIR	
						0 - 300	300-500	500-800	800+	ENTER	LEAVE	BEFORE SHAKE	AFTER SHAKE
1.	HEADINGS -	DRILLING	58	112(112)	176	93	14	5	0	90	143		
2.	-	LOADING	43	55(55)	282	38	9	5	2	110	207		
3	RAISES -	DRILLING											
4.	-	SCRAPING											
5.	STOPES -	DRILLING	58	75(75)	199	61	8	3	3	128	144		
6	-	SCRAPING LHD	35	38(38)	243	28	5	4	0	140	261		
7	-	TIMBERING											
8	-	FILLING											
9	SHAFTS -	DRILLING											
10.	-	LOADING											
11	DUMPING AT GRIZZLIES		9	28(35)	217	20	5	2	1	114	244		
12.	PULLING CHUTES IN BELTS		5	9(5)	297	5	2	2	0				
13	MUCKING - DRAW POINTS												
14	- SCRAMS												
15	SKIPTENDERS BOOTH		1	9(1)	55	9	0	0	0				
16	UNDERGROUND CRUSHERS		1	6(3)	499	2	2		2				
17	UNDERGROUND CONVEYORS		10	41(15)	270	29	4	5	3				
18.	SHAFT LOADING POCKETS		2	18(1)	604	4	4	4	6				
19	FRESH AIR SPLITS		3	7(0)	57	7	0	0	0				
20.	RETURN AIR SPLITS		6	13(0)	80	13	0	0	0				
21.	SHAFTS AND STATIONS		11	19(36)	114	17	2	0	0				
22	MAIN DRIFTS AND X-CUTS		61	102(130)	123	96	3	2	1				
23	TOTALS		303	532(507)	187	422	59	33	18	116	200		

(Source: Personal Communication - Denison Mines)

Knight and Cochrane (174) measured respirable, combustible and mineral dust at Elliot Lake at various points in the operation. Their findings are given in Table 90.

TABLE 90

Composition of the Respirable Dust Produced During Some Typical Mining Operations

Operation	Combustible Dust (%)	Mineral Dust (%)
Jackleg drilling	90	10
Mucking	80	20
LHD	70	30
Slushing	10	90
Chute pulling	50	50
Crushing	ND*	100
Ore and waste passes	ND*	100

(*) ND - not detectable

(Source: Reference 174)

It is clear that the respirable dust varies widely from place to place in the mine in its relative content of mineral and non-mineral (combustible) dust. The combustible portion is probably composed in large measure of diesel soot particulate, except during drilling, where oil may be a significant contributor.

Measurements taken in Zambia in 1972 found a total particulate load of 4.4 mg/m^3 (0.5 - 5.0 u) at a diesel loading operation. Diesel soot was found to make up 80% of the particulate with oils and silica contributing 15% and 8%, respectively. At a drilling site, the total particulate concentration was 2.13 mg/m^3 and was composed of 19% diesel soot, 64% oil and 18% silica. Whether these values are comparable to various Canadian operations requires investigation. Also, no details of the Zambia report were available and the validity of the data needs verification.

3.9.7 Radiation Levels and Ventilation

Since the radon daughter levels in uranium mines are a function of the radon emanation rate and mine air transit times, the ventilation rate and airflow characteristics in such mines is of importance. The shorter the air transit time, the lower the radon daughter concentrations.

Considerable data on the radon daughter levels in the heavily-dieselized Denison Mines are available. However, it is of little value to review the material in view of the substantial increases in ventilation at Denison (see Section 3.9.6) since the time the measurements were taken.

Except for an excursion in 1977, that radon levels have been steadily declining at Denison Mines (as ventilation increased), can be seen in Figure 87. The most recent radiation values available to the authors, provided by Denison Mines were from their semi-annual survey ending September 30, 1977, and can be reviewed in Table 91.

Of the 9,516 samples taken, 8,190, or 86.1%, had a radon daughter content of 0.5 WL or greater.

According to the data in a paper given by Denison personnel in 1976 (150), the group mean radiation exposure in July 1975 was 0.28 WLM, which was reduced to 0.16 WLM in January, 1976.

It has been recommended that the use of tracer gases be used, that particularly in uranium mines where ventilation is critical to radon control. Thimone and Kissell (175) used sulphur hexafluoride (SF_6) to measure air flows which simplified transit time estimations. In addition, the SF_6 was useful in measuring air exchange in areas of low or no air flow, as well as estimating air recirculation due to leakage across solid stopes (176).

A calculation can be made which provides an estimate of the possible extent of attachment of radon and radon daughters between diesel soot particulate, respirable and non-respirable ore dust in ambient mine air.

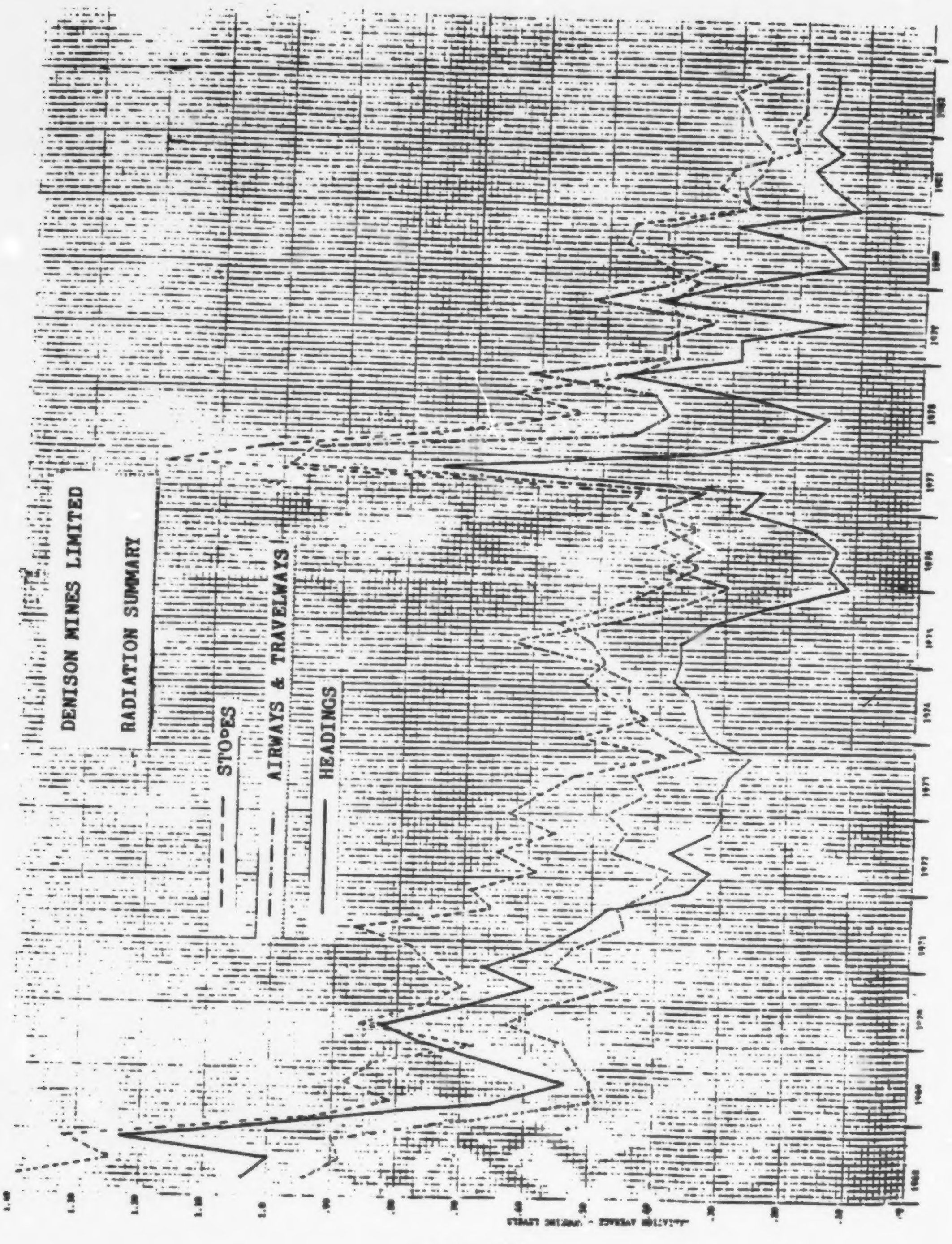

DENISON MINES LIMITED
RADIATION SUMMARY
STOPES
AIRWAYS & TRAVELWAYS
HEADINGS

FIGURE 87

TABLE 91: Analysis of Radioactivity Samples

Mine: DENISON #1 SHAFT

Period Ending: Sept 30/83
1st, 2nd, 3rd Quarters

Location	Total No. of Samples	Number of Samples in Each Range in Working Levels							
		0-0.17	0.18-0.33	0.34-0.49	0.50-0.65	0.66-1.00	1.01-1.50	1.51-3.00	3.00 and over
Headings	312	143	133	26	7	2	1	--	--
Stopes	722	227	209	99	66	66	49	6	--
Travelways & Airways	649	247	130	75	43	63	77	14	--
Misc. U/G Locations	341	201	56	12	17	27	21	7	--
Air Leaving Mine	46	19	5	11	9	1	--	1	--
Leaching Area	70	12	7	8	9	20	12	2	--
Crushing Plant	--	--	--	--	--	--	--	--	--
Mill	--	--	--	--	--	--	--	--	--
TOTAL	2140	849	540	231	151	179	160	30	--

Mine: DENISON #2 SHAFT

Location	Total No. of Samples	0-0.17	0.18-0.33	0.34-0.49	0.50-0.65	0.66-1.00	1.01-1.50	1.51-3.00	3.00 and over
Headings	1574	993	456	90	21	13	1	--	--
Stopes	2259	1006	845	216	137	46	9	--	--
Travelways & Airways	1944	881	517	254	165	97	28	2	--
Misc. U/G Locations	1119	537	288	138	99	44	11	2	--
Air Leaving Mine	42	19	14	4	5	--	--	--	--
Leaching Area	70	5	13	18	10	11	4	7	2
Crushing Plant	--	--	--	--	--	--	--	--	--
Mill	--	--	--	--	--	--	--	--	--
TOTAL	7008	3441	2133	720	437	211	53	11	2

TABLE 91: Analysis of Radioactivity Samples

Mine: DENISON #3 SHAFT

Period Ending: Sept 30/83
1st, 2nd, 3rd Quarters

Location	Total No. of Samples	Number of Samples in Each Range in Working Levels							
		0-0.17	0.18-0.33	0.34-0.49	0.50-0.65	0.66-1.00	1.01-1.50	1.51-3.00	3.00 and over
Headings	96	45	25	9	5	6	3	3	--
Stopes	--	--	--	--	--	--	--	--	--
Travelways & Airways	149	68	37	7	11	14	8	4	--
Misc. U/G Locations	155	46	22	17	16	31	18	4	--
Air Leaving Mine	--	--	--	--	--	--	--	--	--
Leaching Area	13	--	--	--	--	2	3	8	--
Crushing Plant	--	--	--	--	--	--	--	--	--
Mill	--	--	--	--	--	--	--	--	
TOTAL	413	159	84	33	32	53	32	19	--

(Source: Personal Communication - Denison Mines)

If it is assumed that the concentration of respirable diesel soot equals 0.6 mg/m^3 of particles with a mean diameter of 0.5 microns and a density of 0.2 g/cc, the number of soot particles per litre of mine air can be calculated as follows:

Volume of one particle $= 4/3\pi\left(\frac{5 \times 10^{-5}}{2}\right)^3 = 6.5 \times 10^{-14}$ cc

Weight of one particle $= 6.5 \times 10^{-14} \times 0.2 \times 10^3 = 1.3 \times 10^{-11}$ mg

Concentration of particles $= 0.6 \times 10^{-3}$ mg/l

Number of diesel soot particles is equal to:

$$\frac{0.6 \times 10^{-3}}{1.3 \times 10^{-11}} = 4.6 \times 10^7/\text{litre}$$

Alternatively, using figures from Kittelson and Dolan (177) where the density of diesel soot was reported as 1 g/cc with a bimodal distribution around 0.02 and 0.1 microns, a particulate level of 1 x 10^7 per litre can be calculated, which is of the same order of magnitude as the above example.

i.e.:

Volume of one particle $= 4/3\,\pi\,(0.1)^3 = 5.2 \times 10^{-4}$ microns

A concentration of 0.6 mg/m^3 is equivalent to:

$$\frac{0.6 \times 10^{-6}}{1} = 0.6 \times 10^{-6}\ \text{cc/litre} = 0.6 \times 10^{-6} \times (10^4)^3$$
$$= 0.6 \times 10^6\ \text{microns}^4/\text{litre}$$

Thus, the number of particles $= \frac{0.6 \times 10^6}{5.2 \times 10^{-4}} = 1 \times 10^7$ per litre.

Assuming that the bulk of the respirable dust has an aerodynamic diameter equivalent to 2.5 microns and a density of 2.3, the representative ore dust particle would be 1.65 microns in diameter. At a concentration of 0.2 mg/m^3, the number of particles calculate to be 37,000/litre.

Similarly, if the non-respirable dust has an average diameter of 5 microns and density of 2.3 and is present at a concentration of the same order as the respirable factor (0.2 mg/m^3), the number of non-respirable particles present would be 1300/litre.

Thompkins (in "Manual on Mine Ventilation") calculated that ~100 pCi of radiation will be derived from:

1.7×10^6 atoms of radon
977 atoms of RaA
8580 atoms of RaB
6310 atoms of RaC

Thus, at 100 pCi/l, there will be 1.7×10^6 atoms of radon and 15,876 atoms of solid daughters (these values are at equilibrium, depending on residence time, and would be considerably lower in a uranium mine).

On a comparative basis, only diesel soot would be present in sufficient quantity to "bind" radon and its daughters, and thus it must be considered as a possible mechanism for the inhalation of radon products into the respiratory tract in a form which is not immediately expelled (as would be the case for free radon gas) from the lungs. Whether or not attached radon and radon daughters present a greater or lesser health risk than unattached forms requires further clarification (see Section 3.6.3). Evidence will be discussed which suggests PNA's could leach off of particulate matter into lipid media. Evidence for a similar phenomena with radon daughters was not reviewed by the present authors but in their view, the concept warrants study.

It is known that radon daughters will "plate out" onto dust and soot particles (131). However, it is not generally thought that radon gas, which is the dominant form of radioactivity in the uranium mine, behaves similarly. No data were found which examined the potential for diesel soot to be influenced by the particle size of the particulate. Carbon black has a particle size of 0.01 - 0.3 um and diesel engine particulate, according to Lipkea and associates (21) "are part of a dynamic system in which particles at any given time range in size from 0.005 to 30 um, or from near molecular to visible in size". Thus, the size of diesel particulate would seem to be compatible with a potential for binding.

It is apparent that there is a potential for radon gas to adsorb to diesel particulate, which may provide a means by which lung retention times for radon gas could be increased. The extent and significance of this possibility is not known and requires investigation.

3.9.8 Acid Mists

Little information was found in the literature on acid mist levels in mines. As was discussed in Section 3.4.9, the formation of both H_2SO_4 and HNO_3 can occur. In particular, the SO_2 emitted by engines equipped with catalytic converters may be emitted into the mine atmosphere as H_2SO_4.

Laboratory work by Novakov et al (178) suggests that oxidation of SO_2 to sulphate is catalyzed by the presence of soot. This is particularly relevant in that particulate sulphate is believed to exist primarily in the form of H_2SO_4 or $(NH_4)SO_3$, or both.

However, Pierson and co-workers (179), in a study of SO_2 emissions from cars and diesel trucks in tunnels, concluded that the SO_2 oxidation on carbon particles did not contribute significantly to the sulphate levels in the tunnels. Adsorption of sulphate to carbonaceous material was considered negligible due to the presence of a coating which did not adsorb SO_2.

Kirk and Stefanich (121) determined the sulphuric acid level of particulate in the Denison uranium mine in Elliot Lake. Because of the sampling location, the values given in Table 92 are considered "worst case" and are not necessarily representative of the overall mine atmosphere. The operation used No. 1 fuel with a sulphur content of 0.22%. The data suggest that there are significant amounts of sulphuric acid in areas of this dieselized mine which is adhered almost entirely to dust particles in the respirable range.

In a later publication, Kirk and Seymour (180) reported sulphuric acid concentrations in the Elliot Lake Denison and Rio Algom mines. Sample collection was carried out on dust samplers using glass fibre filters and silver membranes over about a 3-hour period. Samples collected on the silver membranes were analyzed for minerals and combustible material by weighing and ashing and for quartz content by x-ray diffraction. Samples collected on the glass fibre filters were analyzed for sulphuric acid content by measurement

TABLE 92

Acid Concentrations in the Mine Environment

	Respirable Dust	Total Dust
[illegible]t (mg/m^3)	0.73	1.07
[illegible]rage H_2SO_4 (mg/m^3)	0.17	0.17
Percent H_2SO_4	23	16

of sulphate ion using a titrimetric technique with a specific-ion electrode with a limit of detection of 0.01 mg/m^3. Respirable and total dust were measured at various points in the heading and near the crusher and provided the data shown in Table 93. Figure 88 gives the maximum concentration of sulphuric acid in the return air as a function of heading air flow. The data suggests that a minimum of 22,500 CFM is required to maintain sulphuric acid levels at 1 mg/m^3 with current mining practices at Elliot Lake. One of the samples in the air return provided a ratio of sulphuric acid in the total dust to that in the respirable dust of 0.73 mg/m^3. This suggested that the sulphuric acid was either remaining as a "free" mist, or, more probably, adhering to the diesel particulate. Table 94 provides a calculation of the sulphuric acid production in mg/ton of ore moved. It was noted that because diesel particulate is extremely hygroscopic the combustible masses given in Table 93 will include varying amounts of water and it is therefore not possible to accurately express the sulphuric acid content as a percentage of the combustible material. However, it was estimated that as much as half of the combustible mass was sulphuric acid. It was suggested that in an ore body with large amounts of silicates, the free silica dust concentration might be the controlling factor in mine ventilation planning, but in a dieselized mine with a low silicate ore control of the sulphuric acid concentration could be a major factor influencing ventilation strategy. The authors concluded that any dust samples collected in an area where diesel equipment is operating should be analyzed for combustible content and approximately 50% of this material, by mass, should be considered as sulphuric acid.

TABLE 93: Measured Dust Concentrations

			Respirable Dust (mg/m^3)				Total Dust (mg/m^3)					
Site	Sampler Location[8]	Airflow kcfm	TRD[1]	RQD[2]	RCD[3]	H_2SO_4[7]	TD[4]	TQ[5]	TC[6]	H_2SO_4[7]	H_2SO_4 Tot. / H_2SO_4 Res.	Production Data
	Underground	--	4.1	2.60	0	0	17.1	5.55	0	0	--	Approx. 800 tons crushed
			3.7	2.31	0	0	12.6	4.89	0	0	--	
	Crusher*		3.8	2.44	0	0	17.1	5.88	0	0	--	
	Intake		0.09	0	0	0.03	0.13	0.02	0	0.04	0.66	53 buckets in 265 minutes sampling time 159 minutes in heading
	Face	35	1.52	0.10	1.36	0.63	1.74	0.17	1.07	0.57	0.27	
1	Scooptram		1.34	0.12	1.20	0.69	1.96	0.19	1.27	0.69	0.28	
	Return 1		1.10	0.07	0.79	0.52	1.71	0.19	1.29	0.58	0.37	
	Return 2		0.87	0.04	0.82	0.29	0.86	0.08	0.79	0.27	0.28	
	Intake		1.35	0.05	0	0	1.10	0.10	0	0	--	20 buckets in 104 minutes sampling time 60 minutes in heading
	Face	22	2.06	0.15	2.30	0.14	2.25	0.29	2.01	0.71	5.07	
2	Scooptram		1.70	0.19	1.98	1.47	2.68	0.28	2.22	1.31	0.89	
	Return 1		2.74	0.19	2.69	0.88	1.92	0.34	1.92	0.87	0.99	
	Return 2		2.50	0.19	2.50	1.11	2.93	0.34	2.64	1.01	0.91	
	Intake		0.97	0.09	1.23	0	1.03	0.14	0.94	0	--	20 buckets in 165 minutes sampling time 100 minutes in heading
	Face	30	2.56	0.35	2.18	0.97	2.65	0.38	2.18	0.76	0.78	
3	Scooptram		2.42	0.38	2.04	0.92	2.88	0.63	2.15	0.85	0.92	
	Return 1		1.34	0.12	1.59	0.42	1.40	0.24	1.10	0.46	1.10	
	Return 2		1.25	0.15	1.59	0.51	1.31	0.24	1.28	0.39	0.76	
	Intake		1.53	0.12	1.24	0.67	1.47	0.18	1.21	0.74	1.10	20 buckets in 170 minutes sampling time 100 minutes in heading
	Face	15	3.83	0.73	2.11	**	4.24	1.78	1.78	1.33	--	
4	Scooptram		2.33	0.40	1.48	1.11	3.01	0.60	1.68	1.14	1.03	
	Return 1		3.97	0.76	2.29	1.73	5.21	1.15	3.26	1.79	1.03	
	Return 2		3.92	0.64	2.25	1.87	1.64	0.96	2.46	1.80	0.96	
	Intake		0.59	0.04	0.74	0	1.51	0.11	1.32	0.98	--	20 buckets in 137 minutes sampling time 85 minutes in heading
	Face	10	3.19	0.26	2.59	1.71	3.22	0.41	2.56	1.30	0.76	
5	Scooptram		2.67	0.22	2.04	1.54	2.74	0.33	2.30	1.40	0.91	
	Return 1		3.35	0.26	2.43	1.51	3.75	0.44	3.09	1.74	1.15	
	Return 2		3.80	0.29	2.99	1.92	3.72	0.44	2.74	**	--	

[1] total respirable dust; [2] respirable quartz dust; [3] respirable combustible dust; [4] total dust; [5] total quartz dust; [6] total combustible dust; [7] sulphuric acid: 0 means less than 0.01; [8] see Fig. 1 for explanation;
*Collected below crusher at air exhaust point; **pump malfunction
(Source: Reference 180)

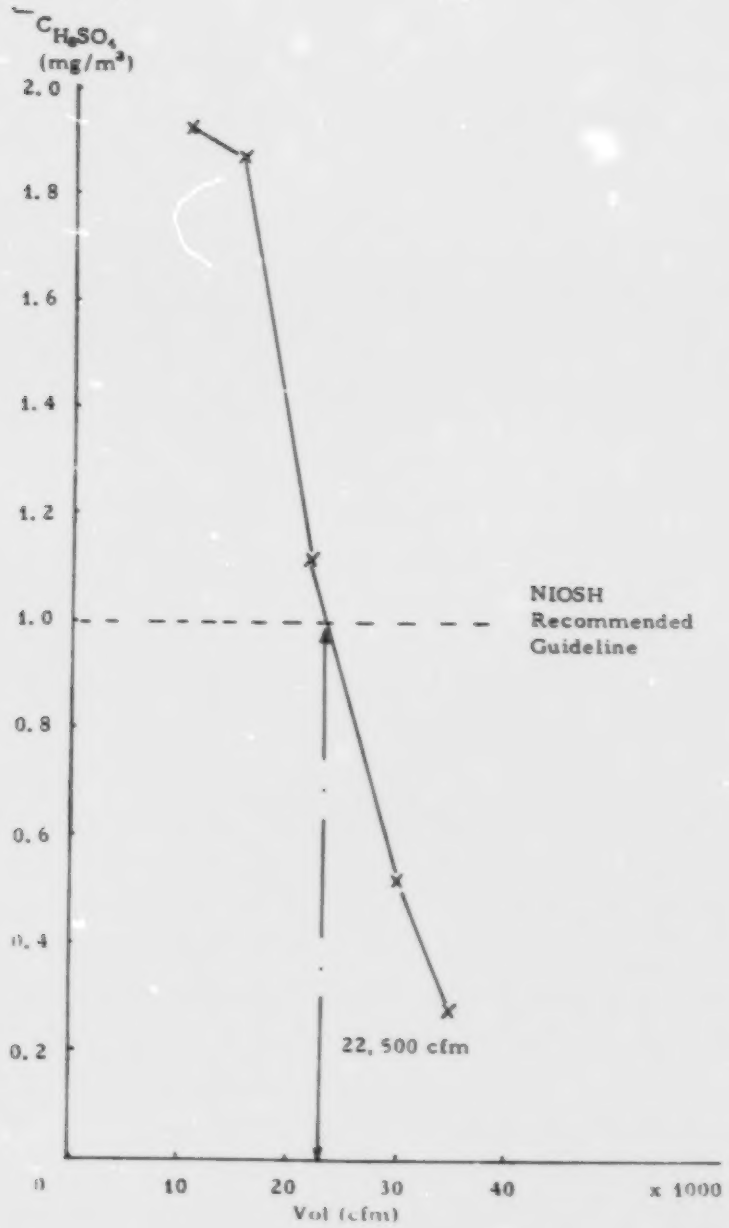


FIGURE 88
H_2SO_4 Concentration Versus Airflow
(Source: Reference 180)

TABLE 94

H_2SO_4 Production (mg/ton)

Site	Respirable Dust	Total Dust
1	182	201
2	206	203
3	223	228
4	281	281
5	227	LOST

(Source: Reference 180)

No other relevant data were reviewed, and thus it is clear that further study of the oxidation of SO_2 and the presence of H_2SO_4 in mine atmospheres is required.

As with other sulphur reactions, if the S content of the fuel is lowered, the significance of this problem will be reduced accordingly. However, the present reality of the situation is that with fuel costs escalating lower sulphur containing fuels are not likely to see increased use.

The potential for adsorbed SO_4 and H_2SO_4 formation to represent a health risk hazard is significant. It must be remembered that although the bulk of fuel sulphur is initially emitted from a diesel engine as free SO_2, some will subsequently be adsorbed onto diesel particulate, and may convert to sulphuric acid or remain as adsorbed SO_2. On catalytic converter-equipped machines some of the SO_2 will be converted to SO_3, and will give rise to sulphuric acid mist directly. This converted and adsorbed SO_2 will not be monitored when ambient SO_2 levels are assessed, but will add to the "combustible particulate" loading, and thereby increase the ventilation requirement, as illustrated by the following example. (The increased ventilation could be regarded as justified as adsorbed SO_2 and sulphuric acid mist are demonstrably more toxic than free SO_2).

This point is illustrated by the following calculation:

An ambient mine atmosphere resulting from the operation of a diesel LHD fueled with 0.25% sulphur fuel could be assumed to contain 0.8 mg/m^3 of soot and 0.75 ppm SO_2 (no SO_2 adsorption or conversion). At these levels, the bracketted term in the additive formula would equal: (The TLV's used are those proposed in the recommendation contained in Section 5.0 of this report: SO_2 = 3 ppm; diesel particulate = 1.5 mg/m^3).

$$\frac{0.75}{3} + \frac{0.8}{2} = (0.25 + 0.40) = 0.65$$

If 25% of the SO_2 or 0.19 ppm should be adsorbed onto the soot it would be assayed as "combustible particulate", increasing that term while reducing the SO_2 value.

Thus: SO_2 (0.75 - 0.19) = 0.56 ppm

and

$$\begin{aligned} 0.19 \text{ ppm } SO_2 &= 0.19 \times 10^{-6} \text{ mg/m}^3 \\ &= 0.19 \times 10^{-6} \times 10^3 \text{ litres/m}^3 \\ &= \frac{0.19 \times 10^{-6} \times 10^3}{22.4} \text{ formula wts/m}^3 \\ &= \frac{0.19 \times 10^{-6} \times 10^3 \times 64}{22.4} \text{ g/m}^3 \\ &= \frac{0.19 \times 10^{-6} \times 10^3 \times 64 \times 10^3}{22.4} = 0.54 \text{ mg/m}^3 \end{aligned}$$

so that "combustible particulate" = 0.8 + 0.54 = 1.3 mg/m^3

The bracketed term now equals:

$$\frac{0.56}{3} + \frac{1.3}{2.0} = (0.19 + 0.65) = 0.84$$

If a further 25% of the SO_2 is converted to sulphuric acid mist (either by the action of a catalytic converter or reaction on the soot surface), the "combustible particulate" would be increased again; and the SO_2 further decreased:

Thus: $SO_2\ (0.75 - 0.19 - 0.19) = 0.37$ ppm

and

$$0.19 \text{ ppm } SO_2 = \frac{0.19 \times 10^{-6} \times 10^{3} \times 64 \times 10^{3}}{22.4} = 0.54 \text{ mg/m}^3$$

so that "combustible particulate" = $0.8 + 0.54 + 0.83 = 2.17$ mg/m^3 and the bracketed term now becomes:

$$\frac{0.37}{3} + \frac{2.17}{2.0} = (0.12 + 1.09) = 1.21$$

These relationships need to be quantitated under actual mine conditions in order to determine whether they are significant or only of academic interest.

3.9.9 Oil Mists

Oil mists are present in underground mine environments and probably derive primarily from the hydraulic systems used in much of the equipment, particularly the jumbo drill rigs. The present authors noted in their visits to mines than thin layers of oil and water quickly accumulated on hats, clothes and faces when standing in proximity to a working face with a three-drill jumbo rig in operation.

Littie quantitative data appear to be available relating to oil mists, and no information was reviewed describing the respirability of oil mists. It may well be that such mists contain a considerable quantity of large non-respirable particles. This is an area which requires study.

The only quantitative information reviewed was from a report at a Mine Ventilation Congress (181) at which it was reported by an attendee from Zambia that the particulate (0.5 - 5.0 u) at a diesel loading site was made up of 78% diesel soot and 15% oil. Values for a drilling operation were essentially reversed with 64% of the total particulate being contributed by oil and 19% by soot. In both instances, the remaining particulate was silica.

Work by Knight and Cochrane (174) suggests that oils may be a significant contributor to the respirable particulate at certain operations. For example, of the respirable particulate present at drilling (jackleg), mucking and LHD operations, the percent combustible dust was 90, 80 and 70%, respectively. In the case of the drilling site in particular, the combustible fraction was probably largely oil as a high diesel soot presence would not be anticipated.

The need for investigation of oil mists takes on added importance if carcinogenic substances, such as nitrosamines or precursors of nitrosamines which could react in the mining atmosphere to form nitrosamines, are present in the oil.

3.9.10 Blasting

It is known that significant quantities of CO, NO, NO_2 and other toxicants are emitted during blasting, particularly when using ammonium nitrate-fuel mixtures, which are in common use in Ontario metal mines (182).

Although a survey of blasting procedures for all Canadian mines was not obtained, it is the authors' understanding that most blasting is carried out during the night shift, when no personnel are in the mines. This is known to be true for at least the Sudbury and Elliot Lake operations. In both instances the men are not permitted to return underground for at least 4 - 5 hours after blasting, during which time any gases released from the blast are swept out of the mine. Under these conditions the blast emissions are not a hazard and need not be considered further.

Measurement of gas levels after blasting by Johnson et al (43) showed that CO was cleared out of the blast area within 1 - 2 hours, which supports the adequacy of the Sudbury and Elliot Lake procedures. These authors concluded that gases from blasting operations were probably moved out of the heading within 2 hours. Some gas could remain trapped in the ore in poorly ventilated stopes, but they would be released when the muck pile was disturbed by the scoop tram. At the very worst, the operator might be exposed to a brief period of moderate gas levels.

Abata et al (183) reported on the toxic gases produced by the detonation of six different explosives in an underground hardrock metal mine during mining operations. The six explosives included two semi-gelatin dynamites, 3 slurry explosives and an ammonium nitrate, fuel-oil/ammonia dynamite combination. Explosives were detonated at the face of the drift and the toxic gases produced by the detonation were trapped at the end of the drift by a bulkhead located a short distance away from the face. It was found that for the 6 explosives tested pollutant levels were within the following ranges:

NO	0.010 - 0.035 ft^3/lb_m
NO_2	0.03 - 0.019 ft^3/lb_m
CO	0.08 - 0.30 ft^3/lb_m
SO_2	0.00 - 0.05 ft^3/lb_m
NH_3	Insufficient data
CO_2	0.7 - 1.8 ft^3/lb_m

In general, toxic gas production seemed to correlate with the densities (kg/m^3) and energy densities (kJ/kg) of the explosives tested.

3.9.11 Nitrosamines

The carcinogenic potential of certain nitrosamines such as dimethylnitrosamine (DMN) has been well established. Some of the most potent animal carcinogens so far studied belong to this group of compounds.

Although detailed references were not located, it has been claimed that in ambient atmospheric conditions, NO and NO_2 in the presence of water can form nitrous acid. Further, nitrous acid can react with secondary amines to form nitrosamines (184, 185). A review of the chemistry of nitrosamine formation can be found in reference (186).

The possibility of spontaneous formation of nitrosamines in urban air has been suggested (185).

Recent measurements with a new instrument designed by Thermo Electron Corporation have found DMN concentrations in urban atmospheres of from 0.02 to 0.8 ug/m^3, depending on the location. Nitrosamines have also been found to be present in cigarette smoke, which can result in the inhalation of 0.8 ug DMN per pack.

The relationship and relevance of these findings to cancer in man are unknown.

Whether nitrosamines could be spontaneously formed in the underground environment is unknown. Oils, which in the past may have contained nitrosamines, are, we understand, presently not formulated with these compounds and therefore oil mists should not be a source. The limiting factor would be the presence of the secondary amines. What their source would be underground is speculative.

Only one instance of nitrosamine measurement in diesel exhaust was found in the literature. Hare et al (58) measured nitrosamines in the exhaust from a GM-DI 6L-71% diesel, as well as a Cummins NTC-290, 4-stroke engine.

No nitrosamines were found in exhaust samples at a detection limit of 0.1 ug on a high-volume filter (particulate weight 0.05 to 0.25 g). In terms of particulate quantity, this limit constituted less than 1 x 10^{-3} percent of particulate in all cases, and less than 1 x 10^{-4} percent of particulate for typical cases.

No data on mine nitrosamine levels were located. In view of the serious potential health effects of nitrosamines and the availability of instrumentation to measure such compounds, it would seem advisable to survey various mine atmospheres for their presence in order to determine the degree of significance of this potential health hazard.

3.9.12 Protective Equipment

No particular effort was put into evaluating protective or filter equipment which could provide miners protection from particulates and other noxious agents. What few articles were reviewed offered little useful information and were mainly discussions of equipment for reducing exposure to dust.

During the authors' tour of Denison Mines, the use of an Airstream Anti-Dust helmet from England (see Figure 2) with a face visor and a rubber face seal, filter and forced (fan-driven) air supply was common with the underground workers. The manufacturer's handbook indicated that when ambient air was passed through the filter, 90% of airborne particulate matter above 0.5 um in size was removed. The filtered air is forced, in a stream, across the worker's face and is contained by the visor and attached face seal. The motor runs off a battery pack and the filters are changed each shift. In speaking with a few men, they indicated that they did not find them objectionable to wear.

Denison Mines indicated that the miners are not required to wear these helmets, but they are made available to them.

Suggestions in the literature have included the use of closed-in cabs on LHD machines, similar to those used on crushers, etc., with provision for filtered air supplies. Obviously, any procedures which reduce the workers' exposure are to be commended. However, economic or technical reasons may limit the usefulness of such techniques. Even more difficult to determine and influence is the desire of the workers to work within or use certain equipment designed to reduce their health risk. In this respect, educational programs may be beneficial and the authors personally feel that it would be appropriate if the mine workers' unions assumed this responsibility.

4.0 HEALTH EFFECTS

4.1 Carbon Dioxide

Carbon dioxide (CO_2) is the end product of oxidation in both living cells and diesel engines. This colourless gas results from the complete combustion of carbonaceous fuel. As oxygen is consumed, CO_2 is produced. If ventilation is inadequate to dissipate the CO_2 produced, its concentration increases and the oxygen concentration falls. Hence, miners who work in poorly-ventilated underground corridors would experience the effect of both lowered oxygen and increased CO_2 if excess CO_2 were present in diesel exhaust.

The problem to be concerned with is the effect of elevated CO_2 in the blood, called hypercarbia (171, 187).

As cellular metabolism produces carbon dioxide it diffuses from the cell into the bloodstream, where it combines with water, producing carbonic acid (H_2CO_3), which dissociates into hydrogen (H^+) and bicarbonate (HCO_3^-) ions. Blood bicarbonate, along with proteins in the blood (such as hemoglobin and plasma proteins), buffer the hydrogen ion to control body acidity and to maintain the blood pH between 7.1 and 7.8. After the blood circulates to the lungs, the CO_2 diffuses across the alveolar membrane and is exhaled into the ambient air. Normally, almost all the CO_2 produced by the body is excreted from it via expiration from the lungs.

If ventilation proves inadequate, the level of CO_2 rises and the pH level of the blood drops when the buffering capacity is exceeded. Respiratory acidosis ensues, and the respiratory centres in the medulla of the brain detect the rising CO_2 level. The rate and depth of respiration increases which increases the expired CO_2 resulting in a return of the CO_2 level towards normal. This ventilatory response is nearly linear when plotted against CO_2 concentrations, as can be seen in Figure 89.

When the respiratory centres in the brain are suppressed, and when the lungs' ventilatory ability is impaired by disease or injury, CO_2 levels build up in the blood (Figure 90) (187).

Carbon dioxide elevation stimulates the respiratory centre with an increased rate of respiration being most marked at 5 percent CO_2 (50,000 ppm) (19). At concentrations of 100,000 ppm, there is asphyxiation and loss of consciousness. However, at low concentrations of 30,000 ppm, CO_2 is only weakly narcotic, but does lead to decreased hearing acuity as well as increased blood pressure and pulse rate, and hence increased work for the heart (171). –

Carbon dioxide is a large proportion of diesel exhaust, ranging from 60,000 to 100,000 ppm (median 70,000 ppm) with the TLV being 5,000 ppm for underground derated diesels (39). The relative dilution to meet the required TLV is 12 to 20 times (171).

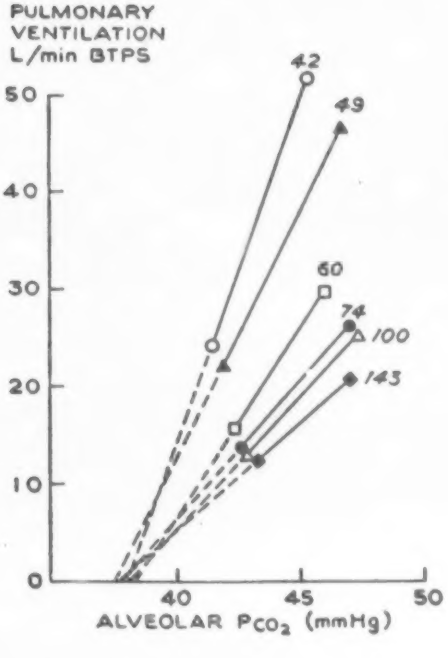


FIGURE 89

Effect of varying degrees of hypoxia on the CO_2 response curve. The number at the top of each line is the alveolar PO_2 during the inhalation of high CO_2 mixtures. The dashed lines do not represent measured values; they are extrapolated from the two measured points.

(Source: Reference 188)

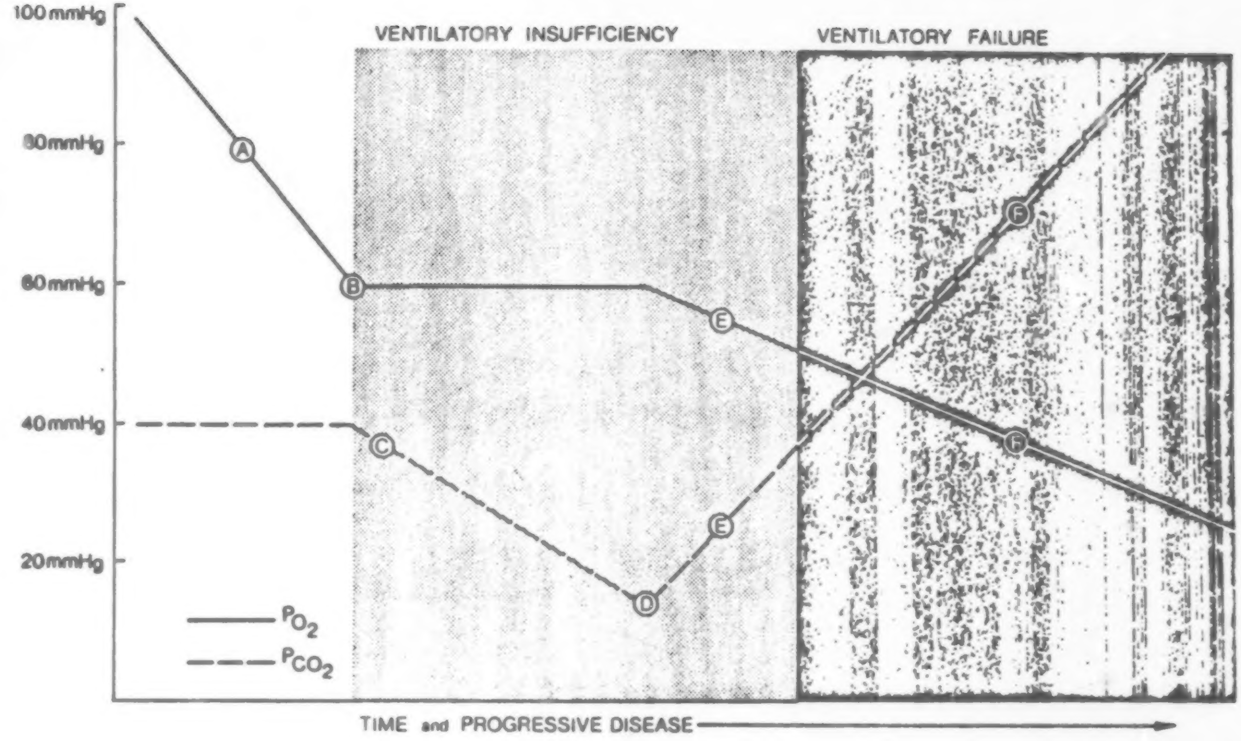


FIGURE 90

Pathogenesis of ventilatory failure. Ventilatory insufficiency - alveolar hyperventilation; ventilatory failure = PCO_2 above normal. A, decreasing arterial PO_2 due to early disease; B, chemoreceptor stimulation begins; C, decreasing PCO_2 in response to chemoreceptor stimulation; D, work of breathing so great that decrease in ventilation begins; E, PO_2 slowly decreases as PCO_2 increases; F, PCO_2 progressively increases while PO_2 progressively decreases.

(Source: Reference 187)

The main concern of occupational health standards is to prevent the level of CO_2 from rising to a value where the oxygen content of the air falls to less than 18 volume % at normal atmospheric pressure (equivalent to 135 mmHg). No serious effects to health or well-being occur at CO_2 concentrations where the O_2 concentration remains at normal levels (171).

Several recent studies show impairment of human behaviour with elevated CO_2 without acidosis. The earliest mental changes show irritability with decreased ability to concentrate. Headache and drowsiness develop as the level of arterial carbon dioxide increases.

In the absence of acidosis, elevated carbon dioxide causes impaired muscle function. This has been shown by decreased exercise tolerance at low concentrations, and heart muscle function is depressed. As CO_2 blood levels increase there is increased cardiac output, blood pressure and blood flow to the brain increases, and there is dilation of brain and heart blood vessels. The direct effect of increased CO_2 is depressant to the heart muscle and the associated acidosis results in the release of adrenalin-like hormones, leading to an increased heart rate, blood pressure and cardiac output.

In animal studies, high levels (30%) of CO_2 were passed for 30 minutes in ambient air to lightly anesthetized mongrel dogs which resulted in an increased rate of respiration, and a marked reduction in renal blood flow and oliguria. No constant change in pH, hematocrit or heart rate was observed. It was postulated that the renal effects observed were the result of a generalized, centrally-induced vasoconstrictor response triggered by inhalation of the high concentration of carbon dioxide (189).

In 1965, Battigelli (190) investigated workers employed on railroad diesels and in diesel repair shops. Exposure of human volunteers for short periods of up to one hour to diesel exhaust at levels comparable to those found in the railroad shops, caused no effects. The CO_2 exposures were up to 11,000 ppm. There was no change in pulmonary function, particularly pulmonary resistance, when the volunteers were exposed to this concentration of diluted exhaust gas for one hour (190).

In an underground coal mine with diesel-powered shuttle cars moving coal to the main return belts, the CO_2 concentration was 675 ppm (67). The exposures of the operators of the continuous miner and roof bolter were found to be the same as that of the shuttle car operator. Equipment duty cycles in this coal mine were averaging 30% (67).

Diesel emission data were collected in the White Pine Copper Mine. Some 200 m^3/minute of air was being exhausted at the face. For a one-hour period, the load-haul-dump machine produced a contaminant concentration at the muck pile in which the CO_2 concentration was 0.225%.

During this sampling period, a CO_2 analyzer mounted on the LHD vehicle continuously recorded CO_2 concentration. Short-term excursions up to 0.49% CO_2 were recorded, suggesting that machine operators can be exposed to up to double the average concentrations at ore loading sites.

The average CO_2 recorded after three weeks measurement on an LHD machine at White Pine Copper Mine was 0.16%. Using the least-square regression lines suggested by Johnson (Section 3.9.4), it can be estimated that the air also contained 4 ppm CO, 12 ppm NO and 1.6 mg/m^3 of respirable particulate (67).

Carbon dioxide, except as a contributor to oxygen deficiency, does not offer serious industrial exposures except in enclosed places where CO_2 poisoning, due to accumulation of higher CO_2 concentrations is always a possibility and which can be fatal (191).

Carbon dioxide levels measured in underground dieselized mine are not sufficiently high to pose a health hazard to exposed miners, even in a poorly ventilated area such as a dead-ended drift.

4.2 Carbon Monoxide

Carbon monoxide (CO) is a ubiquitous gas and pollutant, which is the product of the incomplete combustion of carbonaceous fuels. The gas is colourless, odourless, has about the same density as air, and diffuses to all spaces with air (23). The health effects of increased CO levels are essentially a result of the reduced oxygen-carrying capacity of the blood.

A series of saturation curves demonstrate the different rates at which hemoglobin becomes saturated with CO. These curves are described for varying atmospheric levels of CO in Figure 91.

Individual susceptibility to CO shows wide variation. The health effect is dependent upon the CO in the blood, the oxygen content of the air breathed, the duration of exposure, the ambient temperature, the oxygen demand involved in the work being performed, the metabolic efficiency of the worker, his health status, genotype and capacity to stand exposure. Studies with worker exposure to CO show that carboxyhemoglobin (COHb) increases from 3- to 6-fold as he moves from rest to heavy work because of the individual's increase in breathing rate (23, 171).

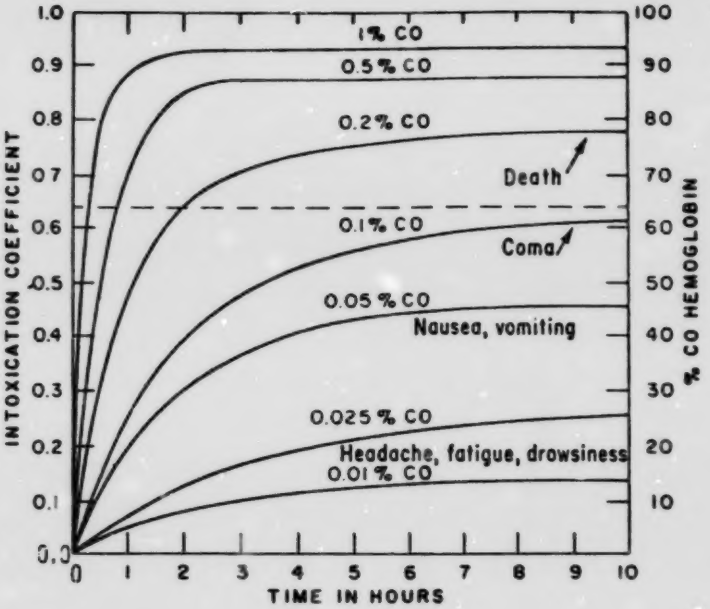


FIGURE 91

Speed of saturation of hemoglobin with different concentrations of CO

(Source: Reference 171)

Variation in individual susceptibility is great; individuals with physiological conditions involving oxygen-carrying capacity, such as anemia, alcoholism, thyroid disease, decreased lung function (including bronchitis), will show increased sensitivity to carbon monoxide (23).

All humans are exposed to some CO. There is endogenous CO production as the body breaks down its own red blood cells in the course of normal function. This process accounts for about 0.4% COHb in human blood. Individuals living in an urban environment develop an additional blood level of 0.6% COHb. Finally, humans who smoke cigarettes or inhale tobacco smoke can have blood levels of 2% to 8% COHb (192).

Carbon monoxide from cigarette smoke and CO in the ambient air are not additive in their biological effect. CO is absorbed only when the partial pressure of CO in the ambient air exceeds that in the pulmonary capillary blood. Thus, persons with COHb levels of 5% from smoking do not absorb further CO from the environment unless the ambient CO concentration is 30 ppm or more. This suggests that smokers may be among the least susceptible persons when exposed to low atmospheric concentrations of CO, since their COHb concentrations are not increased by the exposure (193).

Many recent studies state that the toxic properties of CO are the result of its combination with hemoglobin (Hb). The taking over of the hemoglobin molecule by CO reduces the oxygen-carrying capacity of the hemoglobin or blood, the only vehicle for oxygen transport in the body. Carbon monoxide can easily displace oxygen on the hemoglobin molecule as the affinity of CO for hemoglobin, compared to that of oxygen, is 210 to 1 (194).

Further physiologic changes are enhanced because the presence of COHb alters the dissociation characteristics of oxyhemoglobin (HbO_2) so that less oxygen (O_2) is available to the tissues (194).

The dissociation curve of COHb in the absence of oxygen has the same sigmoid shape as that of oxyhemoglobin, and can be made to coincide with it by multiplying the partial pressure of carbon monoxide coordinates by an affinity constant, as depicted in Figure 92.

Although the shape of the hemoglobin dissociation curve of either oxygen or CO alone is sigmoid, the combination of part of the available hemoglobin with CO shifts the oxygen dissociation curve for the remaining hemoglobin upward and to the left. Examples of this are shown in Figure 93.

These curves illustrate that CO interferes with oxygen transport by two mechanisms: by displacement of oxygen from hemoglobin in the arterial

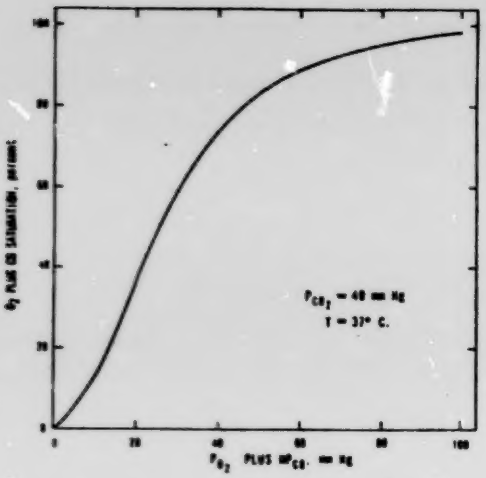


FIGURE 92

Oxyhemoglobin and carboxyhemoglobin dissociation curve. The relative affinity constant (M) is numerically equal to 210.

(Source: Reference 193)

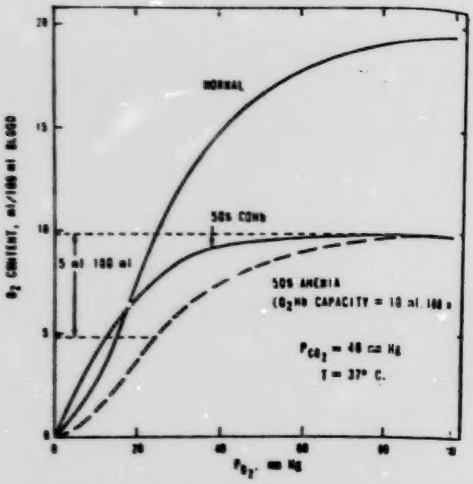


FIGURE 93

Oxyhemoglobin dissociation curves of normal human blood, of blood containing 50% carboxyhemoglobin, and of blood containing 50% of the normal amount of hemoglobin due to anemia. Note that the carboxyhemoglobin curve lies above and to the left of that of blood with an "equivalent" degree of anemia.

(Source: Reference 193)

blood; and by interference with oxygen release in the tissues through alteration of the shape of the oxyhemoglobin dissociation curve (193).

Investigations have shown a close correlation between the percentage of blood saturation and certain signs and symptoms of toxicity. Typical examples are listed in Table 95.

TABLE 95

Signs and Symptoms of Carbon Monoxide Poisoning Correlated with Percentage of Blood Saturation

Signs and Symptoms	Percent of Blood Saturation
None	0 - 10
Tightness across forehead; possibly slight headache, dilation of cutaneous blood	10 - 20
Headache; throbbing in temples	20 - 30
Severe headache, weakness, dizziness, dimness of vision, nausea and vomiting, collapse	30 - 40
Same as previous item but with greater possibility of collapse or syncope; increased respiration and pulse	40 - 50
Syncope, increased respiration and pulse; coma with intermittent convulsions, Cheyne-Stokes respiration	50 - 60
Coma with intermittent convulsions, depressed heart action and respiration, possible death	60 - 70
Weak pulse and slowed respiration; respiratory failure and death	70 - 80

(Source: Reference 194)

The toxic effects of CO are reported to be due to the reduction of the oxygen-carrying capacity of the blood. However, many persons with less than 50% of the normal hemoglobin show no similar toxic symptoms. The explanation is that these persons adjust in some way to the gradually lower hemoglobin levels and can maintain sufficient oxygen tension in the blood. A recent study shows that CO toxicity must have an explanation other than its combination with hemoglobin. It is known that CO competes with oxygen for other heme proteins such as myoglobin, peroxidase, catalases and cytochromes. The mitochondria, where the cytochromes are located, are the site of cellular respiration. Carbon monoxide inhibits cytochrome a_3, an important link in the enzyme system of respiration. Therefore, it is apparent that if CO interferes with cell respiration, toxic symptoms may occur (194).

In order to affect cellular respiration CO must dissolve in the plasma of blood and enter the organs in the same manner as oxygen. In this way, CO competes with oxygen for cytochrome a_3.

A study was undertaken by Goldbaum et al in 1977 (194), to determine the concentration of dissolved CO in blood containing COHb. High concentrations of COHb were associated in blood with minute levels of dissolved CO. Hence, if dissolved CO is the cause of CO toxicity, then high concentrations of COHb in the blood should not necessarily by toxic.

In further studies by Goldbaum et al (194, 195), dogs were transfused with red blood cells with high levels of COHb. Another group of dogs received CO injected into the abdomen. Dogs inhaling 13% CO in air for 15 minutes, died in one hour with average COHb levels of 65%. The dogs injected and transfused, as well as having been made anemic, developed average COHb of about 50%, but did not develop signs or symptoms of CO toxicity.

Although the COHb was circulating at high levels, there was only a low level of dissolved CO in the blood plasma competing with the relatively high concentration of oxygen for the cellular cytochromes. Thus, Goldbaum reasoned that, although COHb was a useful clinical measure of CO exposure, the most important mechanism by which CO caused toxicity was its combination with cytochrome oxidase (194).

Studies in the 1940's, 1950's and 1960's suggested that some psychomotor functions could be disrupted at COHb levels below 5%. However, no clinical manifestations of CO exposure have been shown to occur below 10% COHb (196). The present maximum allowable industrial atmospheric CO concentration is 50 ppm for an 8-hour exposure, which is sufficient to produce COHb levels between 3 and 8%.

An important study in 1963 showed dramatic results in a large number of subjects with COHb levels from 1 to 20%. The results suggested humans could not carry out potentially dangerous or complex tasks after exposure to as little as 50 ppm of CO for 3 hours. The question of cigarette smokers arose since moderate smokers have consistant COHb levels of about 6% (196).

In this study, it was claimed that visual threshold, flicker fusion frequency, time perception and the performance of various psychomotor tasks were altered by COHb concentrations in the 2 to 5% range. The significance of these findings in terms of health or effect on an individual's usual activities was unknown (193, 197).

These disturbing findings led to further studies by other investigators, who noted few changes in psychomotor performance related to carbon monoxide exposure. Only after COHb levels exceeded 15% were physical symptoms, EEG changes and impairment of manual coordination observed.

In the most recent study, people exposed for whole nights in chambers filled with 0, 75 or 150 ppm CO, demonstrated totally negative results in performance. A very subtle effect on sleep patterns was noted; there was less light sleep and more deep sleep in the first few hours of CO exposure.

The authors concluded that at present there was no reason to believe that human psychomotor performance is compromised in healthy humans by exposure to 50 ppm CO for up to 8 hours. This does not encompass possible interaction between this level of CO and other contaminants usually covered by the general term pollution. In this situation, more effects may be documented in the future (193, 198).

Another concern has been the effect of CO on pulmonary function. In a study by Fisher et al (199) in 1969, four human subjects inhaled 6% CO for eighteen seconds with no significant changes in lung volumes, mechanical properties or diffusing capacity.

However, studies have shown pathological changes in the ultrastructure of terminal airway cells in animals. The effects of CO in mice were studied by exposing mice for 17 hours, for 48 hours or 5 days to 50 - 90 ppm CO for varying periods of time, resulting in COHb concentrations of less than 10% in all animals. The terminal bronchiolar and alveolar cells were normal in the control mice, but in the animals exposed to CO progressive changes in the non-ciliated bronchial cells were observed (200).

Changes included an increased number of cristae in the mitochondria, and a dilation of the smooth endoplasmic reticulum filled with a flocculent material. The significance of these changes were not discussed, but the author concluded that concentrations of CO frequently encountered in ambient air (50 ppm and more) may have effects on the fine structure of cells in the terminal airways (200).

The effects of concentrations of CO gas on phagocytic function of guinea pig alveolar macrophages were studied. Impairment of phagocytosis was observed and it was postulated that this may explain the higher incidence of pulmonary disease in urban dwellers who are chronically exposed to automobile and industrial air pollutants (201).

Penney et al (202) noted that data on the cardiovascular effects of prolonged CO exposure to animals and humans is relatively meager. The authors described experiments in which male rats two months of age were continuously exposed to CO concentrations of 100, 200 or 500 ppm. These levels are greatly in excess of those to be found in dieselized mines and therefore have little relevance except to emphasize the probable lack of a health effect of CO levels as found in the working mine environment. For example, after exposure to 100 ppm of CO, the carboxyhemoglobin rose from 0.6% in control animals to 9.3% and the hemoglobin from 15.62 to 16.56%. Although this latter difference was small, it was statistically significant. There were no statistical differences in the body or heart weights of the treated animals. It was suggested that a CO concentration near or slightly below 100 ppm represented the threshold for measurable polycythemic effects in animals exposed continuously over a period of several weeks. One hundred ppm CO was below the threshold for causing measurable cardiac enlargement.

Carbon monoxide has shown a deterimental effect on humans suffering oxygen shortage due to partial blockage of arteries to the heart. Many studies have been done in recent years, such as that of Goldsmith and Aranow in 1975, in which blood levels of CO were measured in smokers and non-smokers (203).

The results showed the concentration of CO in non-smokers to be 0.8 ppm, 0.9 ppm in smokers who have not smoked cigarettes during that day, and 3.8 ppm in pipe and cigar smokers. Those who smoked less than 5 cigarettes over an hour prior to CO measurement had CO levels from 0.4 to 22.4 ppm, with a median of 11.4 ppm; if 5 cigarettes or more were smoked over the previous hour, the CO measured 3.9 to 24.5 ppm, with a median of 14.2 ppm; if less than 5 cigarettes were smoked less than an hour before, 8.5 to 29.0 ppm CO was measured with a median of 18.7; and if a person smoked 56 or more cigarettes less than an hour prior to assay, the CO concentration was determined to be 4.0 to 31.8 ppm, with a median of 17.9 ppm.

The conclusions of this study were: CO aggravates angina pectoris following exposure to 50 ppm of CO after 90 minutes; CO aggravates intermittent claudication after exposure to 50 ppm of CO for 120 minutes; CO causes alterations of the ECG in normal subjects probably following an exposure to 100 ppm after 4 hours; CO produces or aggravates atherosclerosis associated with CO in smoking; and CO impairs survival in patients with acute myocardial infarction. The evidence presented was only suggestive (203).

Patients with diagnosed angina pectoris exposed to CO sufficient to produce a COHb of approximately 5% experienced pain during exercise earlier and for a longer duration than when not exposed. Normal young males and normal middle-aged males exposed to 100 ppm of CO (COHb - 5-9%) were exercised on a treadmill during ECG monitoring and tested for several cardiac functions. Statistical differences were observed only in the older group of subjects. The investigators stated that "...low-level CO exposure may augment the production of exercise-induced myocardial ischemia (deficiency of blood) in persons with pre-existing subclinical heart disease, contribute to the development of myocardial dysfunction, and may lead to an increased incidence of arrhythmias in such persons." (168).

There are many miners aged 45 years or older who may have mild to moderate coronary artery disease. Hence, the studies of Goldsmith and Aranow suggest that these diseased miners may have increased angina pectoris or aggravated coronary artery disease in the mine atmosphere containing the usual levels of carbon monoxide.

In spite of the low levels generally observed in the vicinity of diesel engines in the underground environment, blood concentrations of less than 10% COHb can still adversely affect hearts with coronary disease, terminal airway mucosal cells, and phagocytic function.

The TLV for CO is 50 ppm, which can give rise to blood levels of 3 to 8% COHb. In some miners at risk, this might influence heart and airways function. However, readings taken in mines indicate CO levels are usually less than 10 ppm (64). These levels might affect intracellular organelles in airway cells, but this change has not been conclusively shown to occur in humans.

Under normal conditions, in properly ventilated mines, carbon monoxide levels are generally well below the existing standards of 50 ppm and are not considered to pose a chronic health problem among healthy, conditioned miners. However, care should be exercised in the management of workers with a predisposition for cardiovascular problems, especially those involved in heavy labor, hot environments and altitudes above 5,000 feet.

4.3 Oxides of Nitrogen

When ambient air is compressed in the presence of high temperature, the components, nitrogen and oxygen, interact to form oxides of nitrogen. The main oxide is nitric oxide (NO), which is an environmental pollutant of occupations such as arc welding, chemical works, printing shops, underground blasting in mines, as well as in diesel engine emissions. In all these venues, nitrogen dioxide (NO_2) is also present and is even more toxic than NO. When diesel exhaust releases NO into the air, it is slowly oxidized by the atmospheric oxygen to NO_2.

In Canada and the United States the threshold limit for nitric oxide is 25 ppm. "This is a time-weighted average concentration for a conventional working day, and this concentration may be exceeded, within specific limits, so long as any exposure to a higher concentration is compensated for by spending an appropriate period during the working day in an atmosphere where the concentration is below the threshold limit" (6). In the case of NO_2, there is a ceiling limit of 5 ppm, which is not to be exceeded at any time. In 1976, NIOSH recommended that the NO_2 ceiling should be lowered to 1 ppm as determined by 15 minutes of sampling time (10).

There are nine atmospheric forms of nitrogen including N_2O, NO, NO_2, N_2O_4, N_2O_3 and N_2O_5 plus ammonium, nitrite and nitrate aerosols, but the pollutants of significance in diesel exhaust are NO and NO_2.

Nitric oxide is a colourless, odourless gas which is only slightly soluble in water. However, nitrogen dioxide is a brown gas with a pungent odour which is much more toxic and irritating than NO. In contact with water, nitric acid is formed from NO_2 and nitrous acid from NO.

The toxic effects of NO and NO_2 produce varied pathophysiologic effects in animals and humans. The main effect of NO results from its combination with hemoglobin to form methemoglobin. As with CO toxicity, methemoglobinemia gives rise to a deficiency in oxygen transport by hemoglobin and hypoxemia develops. Nitrogen oxide is not an irritant gas, and as a result, there are no signs or symptoms of the respiratory tract being locally affected (204).

On the other hand, NO_2 is an irritating gas that causes tears, edema and irritation of the nasal mucosa, as well as constriction of the trachea with changes in respiration and pulmonary function tests at levels of 10 to 20 ppm (205) and below (Table 96). There are a myriad of side effects including coughing, shortness of breath, headache, sore throat, nausea and vomiting with permanent changes in lung structure such as pulmonary edema, bronchiolitis obliterans and emphysema with the highest levels of tolerated NO_2 (206)

The cilia in the mucociliary escalator of the airways are affected by the presence of NO_2. Ciliary activity is slowed down which results in stagnation of the mucous blanket in direct proportion to the length of exposure (207). Detailed ultrastructural examination of lung sections in animals exposed to 2 ± 1 ppm NO_2 revealed a loss of cilia, exfoliation of surface cells lining the airways and cellular blebbing (208).

TABLE 96: Toxicologic Effects of Nitrogen Dioxide at Low Concentrations*

Species	Conc. (ppm)	Duration of Exposure	Effect	Comment
Man	5	10 min.	Increased airway resistance in 5/5 subjects. Mean increase 92%.	Response was delayed. Maximal increase 30 min after exposure. Time taken to return to normal not investigated.
Rabbit	0.25	6 h/day	Alteration in structure of lung collagen (by electron microscopy)	Change still apparent in animal sacrificed 7 days after exposure.
Rabbit	1	1 h	Alteration in lung collagen and elastin (by spectroscopy) of animals sacrificed immediately after exposure. Reduced effect observed in animal sacrificed 24 hours after exposure.	Chemical changes suggest denaturation of structural protein only partially reversed.
Rats	0.5 or 1	6 h 1 h	Morphologic change in lung mast cells and release of granular substance in animals sacrificed immediately after exposure.	Effect was partially reversed in 24 - 27 h post-exposure - changes are indicative of inflammation.
Rats	1	4 h	Lipoperoxidation of lung lipids.	Effect delayed. Maximum response occurred 24 - 48 h after exposure.
Rats	2	Cont. for natural lifetime	Pathological changes in bronchiolar epithelium (by electron microscopy) loss of cilia, reduction in normal cellular activity (blebbing).	Changes indicative of a pre-emphysematous condition. Exposure to 0.8 ppm indicates similar process.
Mice	0.5	6-24 h/day for 3 wks	Distended alveoli, loss of cilia, changes in bronchiolar epithelium.	Indicative of emphysematous condition.
		Cont. for 3 months	Increased susceptibility to bacterial pneumonia.	

(*) By volume.

(Source: Reference 205)

As outlined in the section on lung defences, NO_2 is a gas with a lower solubility in water than NO, and hence tends to penetrate further down the airways reaching the alveoli and the respiratory membranes (20). After being inhaled into the deeper parts of the lung, NO_2 can react with water of the alveolar membrane to form nitric acid leading to pathologic lesions in the lung. Irreparable damage to the respiratory membrane occurs and often the only indication to the victim in the early period of damage is a chest x-ray showing small nodules throughout the lungs (209).

The ciliary activity of excised rabbit trachea was arrested by 500 to 1,000 ppm of NH_3, 150 to 200 ppm of NO_2 and 20 to 30 ppm of SO_2 (210). The authors attempted to relate the toxicity of the pulmonary irritant gasses with their resorption into the mucous layers of the respiratory passages. Although data were not conclusive, it was suggested that a gas which rapidly affects ciliary activity in low concentrations and was minimally resorbed in the nasal cavity, would be more toxic than a gas which was largely resorbed in the nose and had little influence on the cilia.

Rats were exposed to 2 ± 1 ppm NO_2 in a controlled atmosphere for their natural lifespans. Animals that had not died by 24 months of exposure were killed and their tissues examined (211). Rats receiving continuous exposure survived their ordinary lifetime with persistent tachypnoea and usually died of non-pulmonary diseases. Resistance to air flow and dynamic compliance did not differ in NO_2-exposed rats compared to controls. Terminal and respiratory bronchiolar epithelium was affected mainly by a loss of exfoliative activity, increased blebbing of cytoplasm in the airways, reduction in or loss of cilia and the appearance of rod-shaped intracytoplasmic crystaloid inclusions. The evidence suggested that the rats exposed to 2 ppm NO_2 had a cleansing function in the periphery of the lung.

In the discussion of the data it was noted that inhaled carbon particles, preceeding or accompanying the breathing of NO_2-containing air, exaggerated the effect of NO_2 by causing destructive lesions in the mouse lungs. This suggested that low ambient levels of NO_2 may be increased in toxicity by concentrating on carbon particles or other aerosol pollutants.

From observations on breathing 1 - 3 ppm NO_2 in air, it was suspected that lesser concentrations would also affect lungs. The conclusion of the study was that "although average ambient concentrations prevailing today are about one order of magnitude lower (i.e. 0.1 - 0.2 ppm), the possibility of deleterious effects in man needs to be kept in mind due to his relatively long lifespan, some 25 to 35 times that of the rat". In addition, in view of man's exposure to NO_2 in smog and tobacco smoke, the ubiquitous gas may possibly contribute to the apparently complex etiology and pathogenesis of emphysema (211).

In monkeys exposed to 10 ppm NO_2, histological examination of lung tissue showed that there were septal breaks in the alveoli and they were markedly expanded. In some areas, large air vesicles were seen with extreme thinning of the septal walls. Moderate expansion of alveoli with minimal wall thinning was also noted in the lung tissue of monkeys exposed to 15 ppm NO_2. Patchy interstitial infiltration with lymphocytes was observed. At concentrations of 15 ppm and above, changes in liver and kidney tissue were also found (212).

Squirrel monkeys exposed to 10 ppm NO_2, and challenged with K. pneumoniae had bacteria present 19 to 51 days after the challenge. In general, K. pneumoniae was not present in the lungs of monkeys challenged with the infectious agent only and killed 15 to 57 days after challenge (212). This suggested that there was an effect of NO_2 on the resistance to respiratory infection in monkeys. Exposure to NO_2 may have permitted a better colonization of bacteria in the lungs of monkeys by inhibition of the mechanism of bacterial clearance from the lungs resulting in increased mortality (212).

Creasia (457) found hamsters pre-exposed daily to 10 ppm NO_2 to develop tolerance to lethal concentrations of NO_2 administered at a later time. However, there was still a proliferation of Type II epithelial cells in the lungs as a repair response to the lung injury resulting from the initial NO_2 exposure. Therefore, the induction of tolerance to NO_2 does not necessarily protect the cell populations of the lung from the cytotoxic effects of NO_2. As with many NO_2 studies the levels used greatly exceed those typical of the mining situation.

From the same laboratory (458), it was found that five hour exposures to extremely high concentrations of NO_2 (40) ppm to rats caused an increase in the synthesis of surfactant-related lipid measured as phosphatidylcholine before the hyperplastic or reparative response of the lung occurred, which was measured as thymidine incorporation into DNA. Ten days following this single exposure there was essentially no difference between the experimental and control groups.

Pickerell et al (459) of the Lovelace Institute, studied the acute effects of NO_2 inhalation in Syrian hamsters. Animals were exposed for 8 hours to 1, 5 or 15 ppm of NO_2 with sacrifice at 1, 2 or 11 days after exposure. Bronchial lavage was completed and differential cell counts and biochemical analyses were made to evaluate the pulmonary cellular effects of the acute exposure. Granulocytes and enriched Type II alveolar pneumocytes were isolated and evaluated for viability and concentration. Airway granulocytes increased one day after exposure to the 15 ppm NO_2 and 2 days after exposure to 5 ppm. The relative fraction of basal cells in the enriched Type II cell population, but not their total cell number, increased at the same time as the granulocytes. Numbers of viable Type II cells recovered were less than 50% of control by one day after exposure to 15 ppm NO_2, by two days after exposure to 5 ppm NO_2 and by 11 days after exposure to 1 ppm NO_2. Palmitate and glycerol incorporation into phosphatidyl choline in the macrophages occurred at the same time as the peak of the viable Type II cells (Figure 94). Incorporation of palmitate into total and unsaturated phospatidylcholine was reduced relative to controls at these times. Conversely at each of the 3 peak response times, incorporation of glycerol into phosphatidylcholine and unsaturated phosphatidylcholine increased. Incorporation of palmitate and glycerol into macrophage lipids increased over 2-fold at 11 days for 1 ppm NO_2. Cell membrane lipid incorporation in Type II cells increased at the same peak response times as for macrophages, although the increase was smallest at the 1 ppm level (Figure 95).

The data demonstrated that exposure for 8 hours to 5 ppm of NO_2 can cause an acute inflammation, as measured by an increase in granulocytes recovered in endobronchial lavage fluid, at the time macrophages were dividing or undergoing repair as indicated by new membrane lipid synthesis. The increased proportion of basalar and ciliated epithelial cells in the "enriched Type II cells" fraction suggested that the inflammation led to upper airway injury. Airway, cellular or lipid incorporation responses peaked at day 1 for 15 ppm NO_2, two days for 5 ppm NO_2 and 11 days for 1 ppm NO_2.

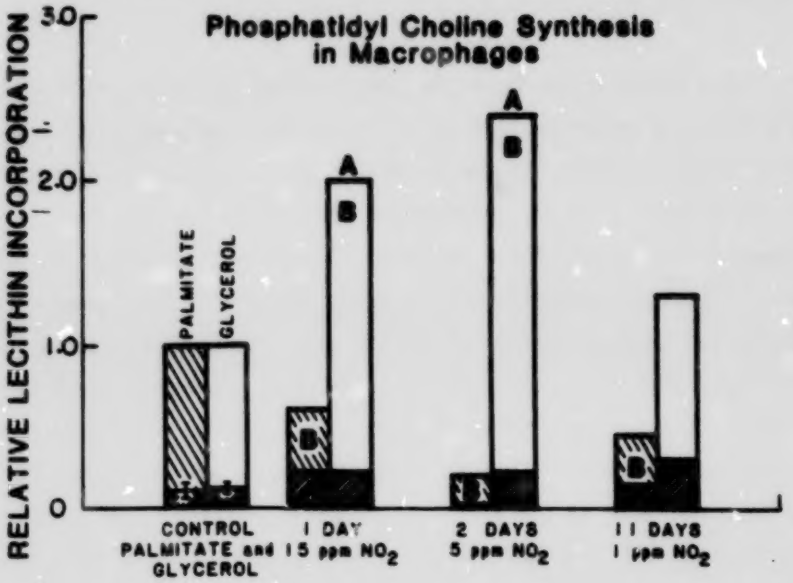


Figure 94 Incorporation of palmitate and glycerol into lecithin in macrophages is shown as a function of ^{3}H-palmitate and ^{14}C glycerol incorporation into the cell fraction relative to control total lecithin incorporation of 1.0, and at peak response times for exposures to 15, 5 and 1 ppm NO_2 for 8 hours. Total lecithin incorporation is represented by the entire column (two crosshatches or clear and crosshatched). Significance ($p \leq 0.05$) is indicated by A. The left column of each pair (two crosshatches) represents palmitate incorporation relative to control hamsters, while the right represents glycerol incorporation (clear and crosshatched). Additional responses are divided into that fraction devoted to cell membrane (unsaturated phosphatidyl choline; upper segment ☐ or ☐) (significance ($p \leq 0.05$) indicated by B), and that devoted to surfactant (disaturated phosphatidyl choline; ☐). Response was normalized to total viable cells present in the incubation mixture. Bars indicate range of incorporation in control hamsters.

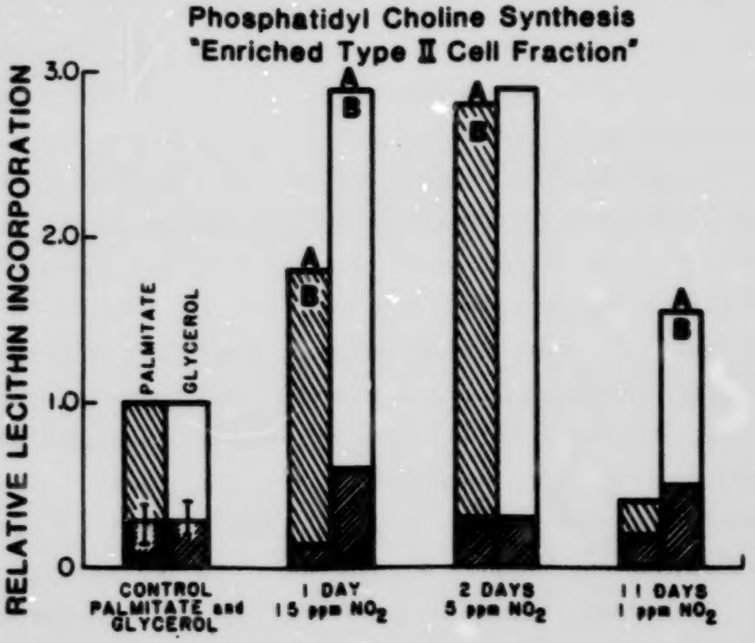


Figure 95 Incorporation into lecithin in the enriched type II cell fraction is shown as a function of ^{3}H-palmitate and ^{14}C glycerol incorporation into this cell fraction. Legends are also shown in figure 1. Response was normalized to total viable cells present in the incubation mixture.

(Source: Reference 459)

In another study from the same group, Johnson, Hahn and Grey (460) found that the response of dogs to inhaled NO_2 appeared to be different from that of smaller animals because of the lack of Type I pneumocyte sloughing and subsequent Type II pneumocyte hyperplasia in the dog. In this study, Beagles were exposed for 6 hours to 70 ppm NO_2 and studied 0.1, 1, 2, 3, 7 and 14 days post-exposure. The gross lesions in the lungs after exposure included excess foam in the airways 0 - 14 days post-exposure, pulmonary air trapping at the same time and increased lung weight 1 - 2 days following exposure. The bronchiolar epithelium was flattened and vacuolated 0 - 7 days after NO_2 administration, with a peak response at two days. The epithelium of alveoli adjacent to the terminal airways contained many damaged Type II pneumocytes and the damage consisted of organelles and cellular swelling, irregular microvilli and loose intracellular junctions. An increase in the number of alveolar pores was present and, in the absence of Type I cell damage, was considered to indicate that poor formation may have resulted from interstitial damage. As man and dog had similar numbers of pores per alveolus, while rodents and most other small animals have significantly fewer pores per alveolus, it was considered the dog might be a suitable model for studying this process.

In additional work from the Lovelace on the subacute effects of low-level nitrogen dioxide exposure to rats, Gregory, Pickerell and Hahn (461) investigated the pulmonary response to 0, 1 or 5 ppm of NO_2 or to 1 ppm NO_2 with two superimposed spikes to 5 ppm NO_2 7 hours per day, five days per week, for up to 2.5 months. Pulmonary cytological, biochemical, histopathological and morphometrical indices were evaluated. Rats exposed to 5 ppm NO_2 had elevated levels of lavage fluid lactic dehydrogenase, but the effect decreased with increasing exposure and by 2.5 months was within normal limits. A similar finding occurred after 8 days of exposure for lung tissue glutathione reductase and at 14 and 30 days for glutathione peroxidase (Figure 96). By the end of the 2.5 month exposure period, all the biochemical endpoints of lung injury measured of lung injury were not significantly different from control values. The lungs of rats exposed to 1 ppm NO_2 were histologically normal, but some animals who received 5 ppm NO_2 and the 1-5 regimen had focal areas of hyperinflation and alveolar macrophage accumulation. In all, subacute inhalation exposure to low levels of NO_2

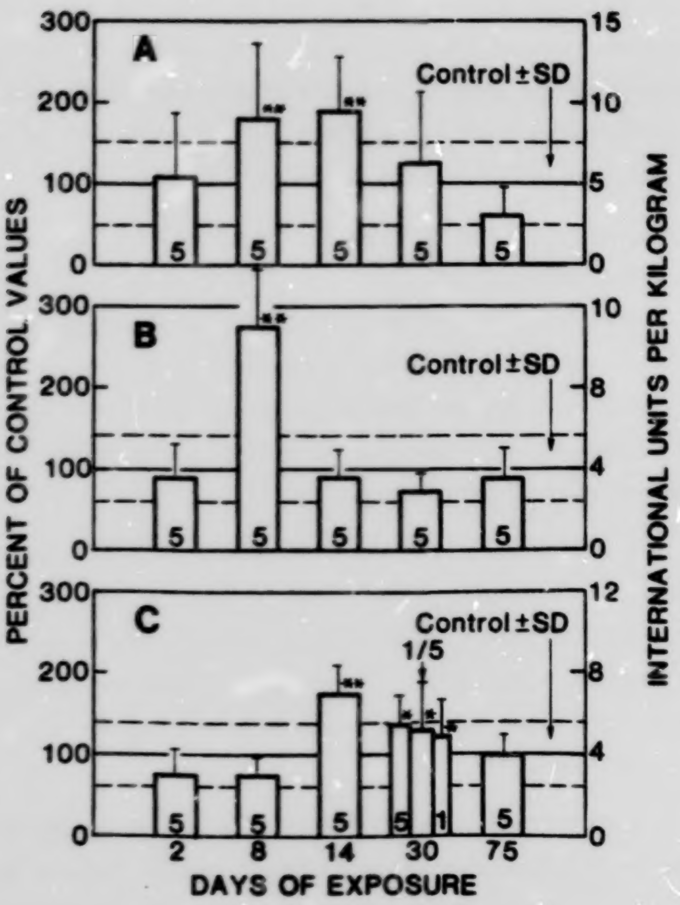


FIGURE 95

Biochemical changes of bronchopulmonary lavage fluid and lung tissue of rats exposed to NO_2 in response to time. Values, percent of control and International Units of enzymatic activity per kilogram of body weight (IU/kg), are expressed as mean ± standard deviation. Statistical significance (* = $p \leq 0.05$ and ** = $p \leq 0.01$) was determined by the nonparametric Mann-Whitney U Statistic. (A) Lavage fluid lactate dehydrogenase. (B) Lung tissue glutathione reductase. (C) Lung tissue glutathione peroxidase.

(Source: Reference 461)

produced little acute lung injury but the trends in the histopatholcgical and morphometrical data suggested development of chronic injury. The investigators were surprised at the unexpected lack of acute lung injury in the 1-5 ppm NO_2 group. It was concluded that exposure to an NO_2 baseline mediates the pulmonary response from the NO_2 spikes. It was concluded that the observed histopathological findings were not present in all exposed animals. However, their presence after 2.5 months of exposure, in the absence of any detectable biochemical injury, suggested the development of chronic lung injury with continued low-level NO_2 exposure.

Animals exposed 6 hours daily, 5 days a week for 18 months to 5 ppm NO_2 showed some enhancement of lung tumours in a susceptible strain of mice (171). It was noted that intermittent exposure of mice to 0.5 ppm NO_2 for 6 or 18 hours per day for 6 months caused a significant increase in the susceptibility to pneumonia and resulted in an increased mortality. However, there was no significant difference between the two experimental groups and the control group after 12 months exposure (213).

Deen and Marinkovic (214) were impressed by a favourable effect of nitrous oxide upon myeloid leukemia and lymphoma colony forming cells. They explored the effect of nitrous oxide on mammary adenocarcinoma development in mice. One group of mice had tumours and received a flow of nitrous oxide of 80%, a second group received 50% oxygen and a third tumour control group was kept in air.

No effect of nitrous oxide at 80% or 50% was observed on the growth of tumours or on the leukocyte count. There was an obvious difference in loss of weight in mice exposed to nitrous oxide, especially at the higher concentrations of NO_2. Consequently, the authors examined the effect of nitrous oxide on tumourous animals receiving chemotherapy. After receiving chemotherapy of various kinds the animals were placed for 20 hours in a nitrous oxide atmosphere. After 20 hours, the animals were sacrified and it was found that the combined therapy produced more weight loss and a higher mortality rate than nitrous oxide alone. Since the tumours of the mice were the same size as those with the mice treated only with cytotoxic drugs, it was concluded that nitrous oxide in combination with cytotoxic drugs did not influence M8013 mammary adenocarcinomas in these particular mice. Nitrous oxide had no inhibitory effect on the growth of M8013 tumours in mice either alone or in combination with cytotoxic drugs.

Hastings et al (215) reported the results of a collaborative study at the University of Cincinnati and the EPA (Cincinnati) to investigate the problems related to sulphuric acid emissions resulting from the use of catalytic converters, which progressed to the study of other selected exhaust components. There was a need to investigate toxic effects that would result from exposure to carbon particulate alone, or to the combination of carbon particulate plus other components, such as NO_2. It was thought that if the gas molecules were absorbed onto respirable carbon particulate, the particulate plus adsorbed gas would reside in the lower lung longer than the gas alone, thus having a greater potential for toxicity.

Weaned rats were placed in running wheels two weeks prior to commencing the test in order to obtain baseline data and then re-examined following exposure.

Every two months pulmonary function and biochemical indices were studied in 10 rats from each group.

No overall activity differences were seen, but there was a significant interaction of treatment with time. Figure 97 indicates that although the control and carbon + NO_2 groups were equal at the outset, they had the greatest separation at 7 weeks. The significance of this finding is questionable in view of the fact that all groups were essentially equal by the end of the 15-week treatment period.

No effect on reproductive qualities were found.

Although there were significant differences in several of the pulmonary function parameters, it was thought premature to interpret them and claim marked differences as a result of exposure. It was concluded that no overt health effects were observed as a result of exposure to NO_2, carbon particulate, or a combination of the two.

Other studies by the same group (215) found a dramatic effect of inhaled aluminum sulphate on the pulmonary alveolar macrophages that resulted in a 3 - 5-fold increase in the leakage of lysosymes into the extracellular fluid. This leakage caused inflammatory changes and resulted in an increase in alveolar wall permeability. Although there was a slight, but significant, increase in extracellular lysozymes, the permeability of the alveolar wall to radio-labelled albumin remained unchanged during the first 4 months of exposure. No effects were seen in the rats exposed to NO_2.

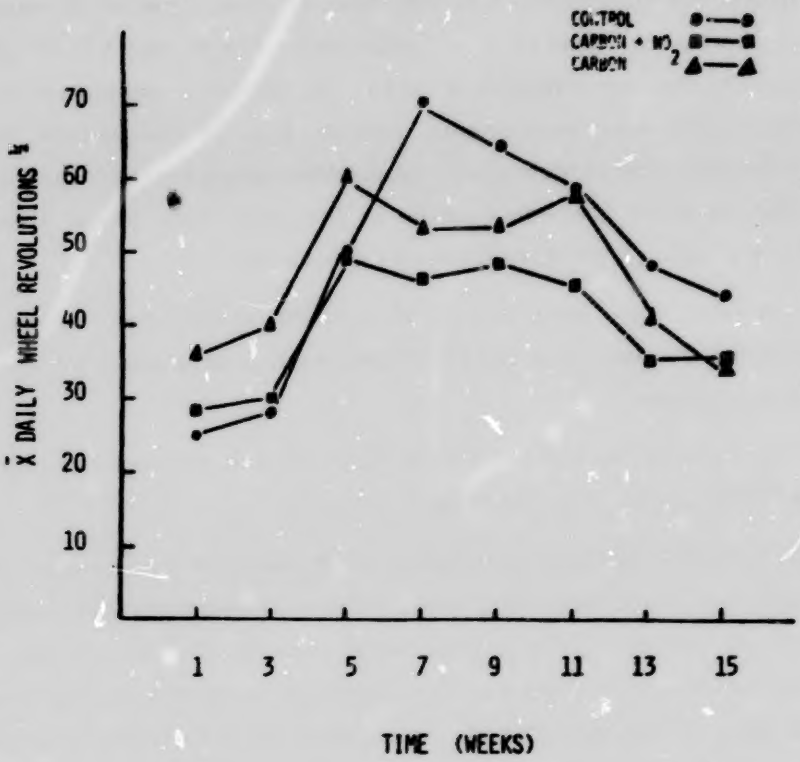


Figure 97

Running Wheel Activity of Rats Exposed to Carbon Particulate Matter Alone, Particulate Matter + NO_2, or Filtered Air. Exposure Began on Week 3 and Continued for the Duration of the Study

(Source: Reference 215)

Although lung pathology was not completed by the time of publication, there were indications that in the NO_2 plus carbon group, a few animals showed dilatation of the peripheral airways and weakening of pulmonary septal walls showing that emphysematous changes were beginning to take place.

From data available at the time of publication no dramatic effects could be attributed to any of the treatment conditions.

The results of pulmonary function tests with exposed and control rats showed that there were no significant differences in mechanical properties, diffusing capacity, the uniformity of distribution, or ventilatory performance.

The animals were exposed a full year for 8 hours per day. The evidence was fairly convincing that the inhalation of diesel exhaust did not result in functional pulmonary alterations in the lungs of rats.

Based on an extensive review of the literature on the oxides of nitrogen, NIOSH concluded that epidemiologic and animal studies which clearly delineate a safe level for human exposure to nitrogen dioxide are not yet available.

Some studies have, however, indicated significant effects on pulmonary function at levels found in mines.

Many studies have been carried out on animals by exposing them to higher levels of NO/NO_2 as well as lifespan low levels of NO/NO_2. However, relatively little is known of the effects of long continuous or intermittent doses of the gases to humans. In 1966, Becklake attempted to study the gaps in knowlege of human exposure to NO_2 and her list of required items of knowledge are given below (216):

1. The effects of long-term, low-dose exposures.
2. The effects of factors operating in combination or in sequence.
3. The effects of agents on enzyme and subcellular levels.
4. The intimate mechanisms of damage by even the simplest substances.
5. The role of adaptation.
6. The role of individual susceptibility.
7. The extremely difficult determination of what constitutes an optimum environment.

Until these effects of NO_2 are known and the human reactions are determined, knowledge of how to control NO/NO_2 and regulate levels in mines will be hampered, and knowledge of the health effects in the human will continue to be vague.

Kerr et al (462) exposed normal subjects and individuals with asthma and chronic bronchitis to 0.5 ppm of NO_2 for 2 hours and measured changes in pulmonary function, and found no significant changes in any pulmonary function measurements in either the healthy individuals or asthmatics, although some of the asthmatic patients did note some increase in chest tightness, running of the eyes, headache and dyspnea with exercise during exposure.

One recent study in Germany involved 298 healthy subjects and 132 patients from a medical institute, age 18 to 72 years. These subjects were given 5 ppm NO_2 or 15 ppm NO over 15 minutes, which caused a significant decrease in the arterial oxygen partial pressure. The diffusion capacity for CO decreased significantly after 15 minutes exposure to 5 ppm NO_2; however, this effect did not show up when NO was inhaled, even when concentrations were raised to 39 ppm (217).

Airways resistance increased significantly after inhalation of NO_2 concentrations of 1.6 to 2.0 ppm or of NO at levels above 20 ppm (217). However, this study was on acute effects in humans at NO/NO_2 levels higher than those usually found in underground mines.

In an underground coal mine where diesel-powered shuttlecars move coal from the mine face to main return belts, the average concentration of NO was found to be 5.3 ppm and NO_2 was 0.5 ppm. In the White Pine Copper Mine, Johnson showed the concentrations around a load-haul-dump unit at the muckpile over one hour were: NO, 19.4 ppm and NO_2, 1 - 2 ppm (64). These are acceptable levels for NO_2 as well as NO, and in view of the current state of knowledge of health effects of NO/NO_2, concentrations such as these should be used as guides for underground exposures at this time.

4.4 Sulphur Oxides

Sulphur dioxide (SO_2) emission from diesels is directly related to the sulphur content of the fuel. The sulphur levels of the various diesel fuels have been commented on. At the time of the 1978 document in Canada, diesel fuel No. 1 with a sulphur content of less than 0.25 percent was the most widely used, especially underground in the Ontario uranium mines (171). However, with rising fuel costs, mines are now using lower grade fuel with a higher sulphur content.

A portion of the SO_2 emitted into the atmosphere undergoes oxidation, leading to the formation of sulphuric acid and particulate sulphates. Sulphur dioxide has a high solubility in water and is largely removed by the surface fluid covering the mucosa of the nasal passages and larger upper airways. Sulphur trioxide affects the lower respiratory tract because its particulate droplet form is carried down into the depths of the airways and alveoli (171).

One study determined the 8-hour lethal concentration (LC_{50}) of sulphuric acid with a mass median diameter (MMD) of 1 micron (u) to be 18 mg/m^3 for one- to two-month-old guinea pigs and 50 mg/m^3 for 18-month-old animals. Other research determined the 8-hour LC_{50} for guinea pigs to be 27 mg/m^3 for 2.7 u particles and 60 mg/m^3 for 0.8 u particles.

Guinea pigs were exposed to 0.1 to 5 ppm SO_2 for periods of up to 12 months. In addition to clinical chemistry tests, pulmonary function of the animals was measured. The average levels of SO_2 used were 0.13, 1.01 and 5.72 ppm. No detrimental changes could be attributed to sulphur dioxide from measurements of tidal volume, respiratory rate, minute volume, dynamic compliance, pulmonary flow resistance or carbon monoxide uptake. Hematology and clinical chemistry measurements were normal. Growth and survival were not adversely affected (218).

Studies have been reported in which guinea pigs were exposed to aerosols of sulphur dioxide (1.1 ± 0.1 ppm) in a polydispersed sodium chloride aerosol (900 to 1000 ug/m^3) and water vapour which were mixed with filtered air to provide a flow of 10 litres/minute. Animals were lightly anesthetized and their pulmonary flow resistance measured. The size distribution of the aerosol was essentially that of urban air, with peak particle counts occurring at 0.1 um and with no particles greater than 2 um (219).

Six modes of exposure were investigated: SO_2, at less than 40% relative humidity (RH); SO_2 at greater than 80% RH; sodium chloride at low RH; sodium chloride at high RH; SO_2 plus sodium chloride at low RH; SO_2 plus sodium chloride at high RH. After a control period, each animal was exposed to one mode for one hour, allowed to recover on ambient filtered air for one hour, and then exposed again to another mode for a second hour. The sequence of modes was random.

Pulmonary flow resistance increased significantly only when SO_2 and NaCl were administered concomitantly at high RH. In certain conditions, particularly NaCl with SO_2 at low and high RH, SO_2 was absorbed by the aerosol which was substantiated by the measuring of a reduction in the pH of the aerosol.

No sulphuric acid was detected using a method in which sulphuric acid was reacted with ammonia to form ammonium sulphate, which was identified by its deliquescence point. Mass spectrometer analyses also indicated the absence of H_2SO_4. Once the RH was sufficiently elevated, the hydration of the particles and their subsequent uptake of SO_2 proceeded rapidly. The nasal passages provide a large moist surface which could absorb large quantities of SO_2 while the particles still attract water vapour.

The results were in agreement with the hypothesis for a gas-aerosol synergism. As far as was known, this was the first demonstration of such synergism in which the physicochemical basis for the biological effect was shown experimentally (219).

Studies up to 1969 had failed to demonstrate changes in lung mechanics (220). However, investigations reported in 1975 have shown pulmonary function changes. The SO_2 concentration in the atmosphere in Ottawa was registered at 16.1 ppb and in Sudbury was 32.5 ppb. Suspended particulate matter was 90.5 and 52.1 ng/m^3, respectively (1971 - 1973). It was pointed out that shut-down of the mining and smelting operations was required in Sudbury when the SO_2 concentration, over a 24-hour period, averaged 0.3 ppm or the particulate was 400 ng/m^3 (221).

Residents from the two locales (3,280 in Ottawa and 2,208 in Sudbury) were put into one of three groups according to smoking history: never smoked, ex-smoker or current smoker. The current smokers were sub-divided into three classes of less than 10 cigarettes per day, 10 cigarettes to one package, and more than one package per day. A mixed class of cigar and pipe smokers was also included.

Cigarette smoking was found to be the dominant influence on disease frequency and lung function was significantly poorer in residents of the more polluted Sudbury area. The FEV/FVC of those examined was lower in Sudbury than in Ottawa. The prevalence of symptomatic bronchitis was significantly higher in Sudbury males than in Ottawa males, but no such difference could be found between females from the two locations (221).

Another study measured, concurrently, the effect of SO_2 on pulmonary function and its concentration and localization in blood and tissues. Ten dogs were exposed to concentrations of radioactive $^{35}SO_2$ in air ranging from 1.8 to 148 ppm for a period of from 30 to 40 minutes. The compliance of the lungs and thorax decreased slightly from the control values by an average of 8.5%, which is statistically significant. There was no direct relationship evident between the amount of decreasing compliance and either the total amount of $^{35}SO_2$ retained or the micrograms of $^{35}SO_2$ retained per pound of body weight.

During exposure to $^{35}SO_2$, there was an increase in the non-elastic resistance to breathing in all instances ranging from 150% to over 300%.

Following the exposure period when these dogs were again breathing air, the non-elastic resistance to breathing returned to the control level, or to within 2 - 3% of this level.

It is interesting to note that the increase in non-elastic resistance occurred within 9 seconds after the onset of breathing $^{35}SO_2$, and disappeared as quickly following the end of exposure.

Sulphur-35 was found to be highest in concentration in the trachea, lungs and hilar lymph nodes, and next highest in the kidneys and esophagus. A relatively uniform and low concentration was noted in other tissues, such as the heart muscle, liver, spleen, and striated muscle. Sulphur dioxide-35 was absorbed quickly, distributed widely and readily gained access to many tissues, including the brain (222).

The functional response of several levels of the respiratory system to the nasal inhalation of SO_2 was investigated. Mongrel dogs were lightly anesthetized and exposed to SO_2 by nose, then by tracheal cannula. An isolated segment of trachea was also exposed. Concentrations of SO_2 ranged from 7 to 230 ppm with an exposure period of 15 to 20 minutes (223).

Nasal flow resistance increased roughly in proportion to the concentration of the gas. This change occurred despite a slight average reduction in flow rate and became progressively greater with time in response to 25 to 61 ppm of SO_2. During recovery, that is after removal from the SO_2, the nasal flow resistance reverted partially or completely to control levels. Pulmonary flow resistance underwent fewer and relatively smaller changes than the nasal flow resistance. The pulmonary flow resistance rose and fell with about equal frequency; however, during recovery from SO_2, the pulmonary flow resistance tended to increase. The changes in laryngeal flow resistance approximated those of the lungs.

In a second set of experiments, in which SO_2 was administered by tracheal cannula, the pulmonary flow resistance usually rose quickly, reaching a peak within several minutes and then decreased. Exposure of this type evoked the greatest change in pulmonary flow resistance observed throughout the study. Limiting the area of exposure to the larynx and upper trachea produced a less pronounced response in the pulmonary flow resistance. The pressure-gradient across the upper laryngotracheal segment, for a constant rate of flow, increased upon direct exposure of the segment to SO_2 (223).

The inhalation of a chemical initiates responses which can be grouped into four areas: the airways which respond by bronchoconstriction to initiate the cough reflex; the bronchial blood vessels which undergo changes to reduce absorption of the chemical substances via the bronchial mucosa; the pulmonary blood vessels which respond to reduce absorption of the toxic irritants via the alveolar capillaries; and the heart and systemic vessels which retard the distribution of the chemical substance to the vital organs.

These mechanisms have been regarded as four successive lines of defence against the absorption of cigarette smoke and are now being discussed with regard to human response to sulphur dioxide and sulphur trioxide. Several reports considered whether any of the responses measured could be regarded as sensitive indicators for the occurrence of the pollutant at dangerous concentrations in an atmosphere.

Following exposure to SO_2, the entire respiratory passage undergoes some functional changes. The most conspicuous response is coughing, but this appears to be the last manifestation of the chain of events originating from the initial contact between the mucosa lining the passages and the SO_2.

Concentrations of 12 to 15 ppm SO_2 are slightly irritating to the nasal mucosa. This concentration is 500 times that which causes irritation of the eyes so that the respiratory system is considerably less sensitive to irritation. The mucosal irritation by SO_2 triggers most of the observed events. In addition, the irritation suppresses the rate of ciliary activity. It is commonly agreed that 15 ppm SO_2 causes nasal irritation and that most of the SO_2 is absorbed by the nasal mucosa. Bronchoconstriction has been reported at concentrations of 28 ppm when inspired intranasally or if lower concentrations (15 ppm) are inspired orally. Bronchoconstriction can occur without any signs of nasal irritation. Following the exposure of the lung to SO_2, the bronchial veins are constricted and most of the blood in the bronchial capillaries is shunted towards the bronchopulmonary vessels.

Concentrations of SO_2 approaching 400 to 6,000 ppm cause pulmonary edema and hemorrhage in cats and rabbits. Exposure to concentrations of SO_2 exceeding 400 ppm causes death within 24 hours in most animal species.

The pulmonary effects resulting from the inhalation of sulphur trioxide resemble those of sulphur dioxide (224).

Transient acute effects of SO_2 have been observed in pulmonary function tests on humans. These changes have been seen even in exposure to SO_2 in the range of 1 to 5 ppm in laboratory studies. Also, epidemiological studies of humans exposed to SO_2 in their work environment suggest that there is an association with chronic non-specific pulmonary disease. However, these studies have failed to define adequately the levels of chronic exposure resulting in deleterious effects (16).

One pilot study investigated both long-term and short-term changes in airways resistance in humans. Environmental conditions with SO_2 were used in that two subjects walked a distance in downtown London, England. This provided repeated exposure over a long period of time to atmospheric pollution and weather conditions, with exercise, prior to making measurements of airway resistance. Combining the results from the two individuals, significant correlations were found between the specific airway resistance measured just after the walk, and the logarithms of daily mean concentrations of smoke, SO_2 and net particulate acid. Increased specific airway resistance was associated with higher levels of pollution and SO_2. It should be noted that the changes in the specific airway resistance demonstrated were not clinically significant in normal persons. Changes in specific airway resistance between a mildly polluted day with SO_2 at an average concentration of 200 ug/m^3 and a day of high SO_2 (1000 ug/m^3), would be from 2.27 to 4.06 cm H_2O/l/sec. The authors emphasized that the effects not have been due solely to sulphur dioxide, but they could conclude that the changes appeared to be more closely related to this gas than to any other variable measured. Although the changes were not clinically significant in normal persons, those with respiratory disease may be affected (225).

Hiller and Bone (463) noted in a review that it is not clear whether there are any significant health effects when levels of SO_2 are within the OSHA 8-hour exposure averages of 13,000 mcg/m^3 (5 ppm) for industrial workplaces. They drew attention to Kreisman and associates (464) who showed reduced flow rates when subjects were exercised while inhaling 3 ppm of SO_2.

Bedi et al (465) administered SO_2 at a level of 0.4 ppm alone and in combination with 0.4 ppm of O_3 to normal volunteers for 2 hours, who exercised 15 minutes every half hour. Pulmonary function was measured including standard spirometry, thoracic gas volumes, airway resistance, closing volumes and maximum voluntary ventilation. No pulmonary function changes occurred during exposure to SO_2 alone.

Pulmonary function of 113 copper smelter workers was measured in 1973 and again in 1974 to assess the effects of chronic exposure to SO_2. Respiratory symptoms, smoking and occupational history, and pulmonary function as measured by forced vital capacity (FVC) and one-second forced expiratory volume (FEV_1) were recorded.

Exposures to SO_2 were in concentrations ranging from 1.6 to 45 ppm. The highest levels in the smelter occurred close to production sources. The smelter workers' SO_2 exposure was divided at less than 2.6 mg/m^3 and equal to or greater than 2.6 mg/m^3. This division was selected on the basis of laboratory work with humans which suggested 1 ppm to be the lower limit at which acute exposures cause respiratory effects (16) (see Figure 98 and Table 97).

Exposure to 1.0 to 2.5 ppm of SO_2 was associated with excessive loss of FEV_1 during the year, and an increase in respiratory symptoms, after adjustment for smoking habits. Workers with an FEV_1 below normal on initial measurements (based on age and height) showed evidence of even greater losses of pulmonary function related to SO_2 exposure (16).

There have been varying reports on increased airways resistance due to changes in environmental conditions, especially with reference to increased SO_2 in the ambient air.

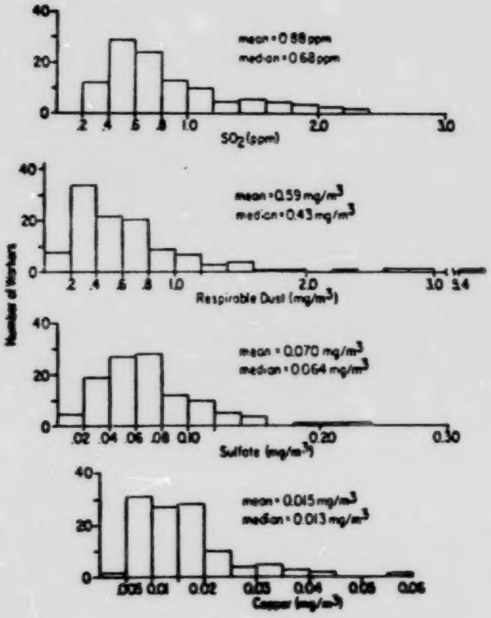


FIGURE 98

Histogram of smelter workers' exposures to SO_2, respirable dust, sulphate and copper

(Source: Reference 16)

TABLE 97

Pulmonary and Personal Characteristics of Smelter Workers*

Variable	Smelter (mean)	Smelter (SD)	Discher's Predicted**	Percent of Predicted
Age in 1973, years	36.0	1.09		
Height, cm	178.0	0.69		
FVC, ml				
1973	4,720	81.5	4,798	98.0
1974	4,680	81.7	4,771	97.7
1973-74 change	-39.8	22.2	-27	152
FEV_1, ml				
1973	3,682	69.5	4,020	91.2
1974	3,628	69.7	3,994	90.4
173-74 change	-54.6	15.1	-26	210

* A total of 113 measurements.

** These values represent the predicted pulmonary function of the smelter workers, using data from healthy industrial workers of the same age and height.

(Source: Reference 16)

In one study, Cynomologous monkeys were exposed for 18 months to concentrations of SO_2 ranging from 0.1 to 5.0 ppm. This range was selected by the researchers because it included those concentrations encountered in urban atmospheres and extended to the current TLV of 5.0 ppm established by the ACGIH. The results indicated that no detrimental changes in monkeys could be attributed to continuous exposure to SO_2 at 0.14, 0.64 or 1.28 ppm for a period of 78 weeks. The group exposed to 4.69 ppm for a period of 30 weeks did not seem to be adversely affected (226).

An accidental over-exposure to SO_2 occurred after 30 weeks of exposure in the chamber in which animals were being exposed to 4.69 ppm. The concentration during the one-hour over-exposure was estimated to be not higher than 1000 ppm and not lower than 200 ppm. The animals were not re-exposed to SO_2, but were kept in the chamber with filtered air for the following 48 weeks. The over-exposure accident was followed by a definite deterioration in pulmonary function, which manifested as changes in the distribution of pulmonary ventilation as measured by the nitrogen wash-out technique. The microscopic findings in this group indicated that not only the lungs had been adversely affected, but also alterations in the liver, similar to those seen in guinea pigs exposed to 5.72 ppm SO_2 were present.

It was concluded that no damaging effects could be attributed to SO_2 under the conditions of the experiment. The results were substantiated by the fact that histopathological examination of the lungs confirmed the observations resulting from pulmonary function tests.

The findings presented in this article, and the results reported in earlier articles using guinea pigs, indicated that SO_2 in concentrations up to 1 ppm did not cause alterations in pulmonary function, nor induce detrimental changes that could be seen on microscopic examination of pulmonary tissue. Also, normal results have been obtained in dogs who had been chronically exposed to low concentrations of SO_2 (226).

However, another study looked into the physiological responses of human subjects on exposure to low levels of SO_2.

In the first series of experiments, subjects were exposed by mouth to SO_2 in concentrations of 1 - 2 ppm, 4 - 7 ppm, and 10 - 16 ppm. Exposures lasted 10 - 30 minutes, and were spaced at least one month apart. Only one subject showed any increase in pulmonary flow resistance in response to 1 - 2 ppm of SO_2. It should be noted that his control pulmonary flow resistance was also the highest. The group, in response to 4 - 7 ppm SO_2 for 10 minutes, showed an average increase in pulmonary flow resistance of 39% above control levels. At the highest concentration, there was a rise in this group of 72% above control. The change in pulmonary flow resistance occurred within the first minute of exposure and was maximal after 5 -10 minutes. Five of the exposures were prolonged for 30 minutes. Although the peak flow rate (PFR) remained elevated during the entire exposure, it diminished somewhat after the first 10 minutes.

The end expiratory thoracic gas volume showed no consistent change when 1 and 5 ppm of SO_2 were administered, and an average increase of only 0.3 litres in response to 13 ppm. An increase in thoracic gas volume would tend to offset the observed change in the PFR.

A discrepancy in timing was observed between the onset and progress of symptoms and the changes in PFR. Coughing, throat and upper chest irritation appeared with the first few breaths of SO_2. The symptoms reached a peak within a few minutes, but thereafter subsided at a time when the mechanical response of the lungs was still intensified (227).

In a further study, changes in PFR were compared when the subjects breathed SO_2 by nose or by mouth. The level of gas was deliberately set high at 15 and 29 ppm, to emphasize possible differences in response. PFR increased significantly in 9 of the 12 experiments in which SO_2 was administered by mouth, whereas an increase was found in only 3 out of 12 exposures to the same levels of gas by nose. The average changes are shown in Figure 99.

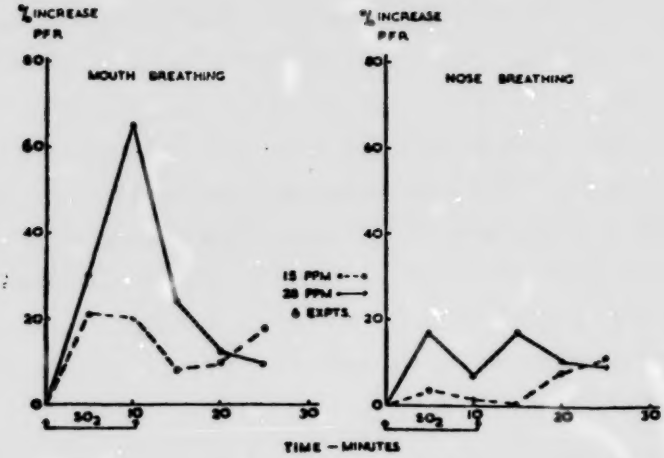


FIGURE 99

Comparison of changes in PFR during exposure to SO_2 by mouth and by nose

(Source: Reference 227)

Although nasal irritation was uncommon, several subjects exhibited an increase in nasal flow resistance (NFR). In fact, in response to 15 ppm of SO_2, NFR rose, on average, more than did PFR. The subjects rarely coughed or experienced chest irritation when breathing SO_2 by nose. These same symptoms were not infrequent when exposure was by mouth (227).

In another set of experiments, SO_2 absorption was measured in 7 subjects exposed to SO_2 by nose. The average concentration of SO_2 entering the nose was 12.4 ppm and of this amount, nearly 99% was absorbed by the nasal pharyngeal mucosa during inspiration. The rate of absorption did not change over the 30-minute period of exposure, and the expired gas coming from the lungs was essentially free of SO_2 at the level of the pharynx. The net uptake by the airways was determined to be 84% (227).

The synergistic effect between particles and vapours has received increasing attention, with respect to air pollutants and the development of pulmonary disease. Early studies in the 1960's demonstrated that concurrent exposure to SO_2 and carbon black particles, small enough to be in the respirable range, caused development of effects not evident when either of the two pollutants were present separately (228, 229).

In one study, guinea pigs were exposed to manganese dioxide (MnO_2) alone and MnO_2 plus SO_2. Viable, non-pathogenic bacteria (E. coli) were inhaled by half the guinea pigs in each group.

The ability of the lung to clear inert particles was found to be decreased in animals exposed to MnO_2 plus SO_2 compared to control animals, or to animals exposed to either of the two agents alone (Table 98). There was a decreased clearance of viable bacteria noted in the same group. This study indicated the synergistic effect between particles and SO_2 in the development of biological effects (229).

The close association between the occurrence of sub-pleural alveolar hyperplasia and neoplasia and the presence of engorged lymphatics filled with apparently normal lymphocytes in the affected sector of the lung has been reported (230). However, it was the experience in one study that sub-pleural alveolar hyperplasia is rarely observed in the absence of lymphatic engorgement. Experiments were devised which would create areas of sub-pleural lymphatic engorgement and maintain them for a known length of time, and the possible effects of such treatment on the subsequent incidence of hyperplastic and neoplastic lesions in the lung observed.

TABLE 98

Killed Radioactive and Viable E. Coli Remaining In Guinea Pig Lungs 3 Hours After Exposure. Expressed as Percent of 0-Hour Value

Treatment	Amount	Radioactive	Viable
Control	--	75	16
MnO_2	5.9 mg/m^3	79	19
SO_2	6 ppm	88	10
MnO_2 + SO_2	5.9 mg/m^3 + 5 ppm	118	28
1969			
Coal	15 mg/m^3	74	43
SO_2	10.4 ppm	64	21
Coal + SO_2	15 mg/m^3 + 10.4 ppm	102	44

(Source: Reference 229)

Rats were divided into three groups: Group 1 consisted of untreated controls; Group 2 was exposed to inhalation of free radicals produced by radio-frequency discharge; and Group 3 was exposed to 500 ppm SO_2 for 5 minutes.

No lung tumours were observed in mice below 300 days of age, and only those which survived this age were considered in assessing the results. An increased incidence of primary lung tumours in both sexes exposed to SO_2 was approximately doubled as compared with controls. Carcinoma of the lung in females was observed only in those exposed to SO_2. There was a slight increase in lung tumours in both sexes exposed to free radicals. The incidence of hepatoma and lymphomatosis, the next most frequent tumour in control mice, was unaffected by the experimental conditions. There was an association between persistent lymphatic engorgement, alveolar hyperplasia and the development of progressive neoplasia, papillary edema, and carcinoma of the lungs of mice in all groups. Repeated exposure to SO_2

apparently accelerated the unexplained sequence of events which lead to the growth of spontaneous lung tumours in the mouse (230).

Sulphur dioxide has been implicated in the stimulation of lung cancer in mice. Peacock and Spence submitted LX mice to 500 ppm of SO_2 five days a week for up to 2 years. There was an increased incidence of primary pulmonary neoplasia up to 54% in males and 43% in females compared to control mice. The tumours were adenomas and primary carcinomas.

It is thought that lung parenchyma engorgement of lymphatics by lymphocytes in immediate juxtaposition to areas of alveolar epithelial hyperplasia, is associated with eventual development of neoplasia (228, 230).

However, no physiological mechanism of action has been uncovered to explain how neoplasia begins and grows in these areas.

In a study of respiratory mechanics, Sim and Pattle (231) exposed volunteers to more than 50 ppm SO_2 for 10 minutes, or to 9 ppm for 60 minutes. One-half the subjects experienced increased airway resistance accompanied by inflammation of the nasal passages, tearing, and abnormal chest sounds. Several investigators have reported increases in airway resistance after exposure to 5 ppm SO_2, but rapid adaptation to these effects occured.

The results of exposure to SO_2 in combination with particulate and other sulphur oxides has been extensively studied by Amdur and her colleagues. They reported that particle size is an important determinant of effect with the smaller particles having a greater effect, and the more soluble the aerosol the greater the potentiation. Soluble metallic aerosols produce greater responses than inert aerosols and the effects of aerosol-SO_2 mixtures persist longer than those of SO_2 alone. Also, aerosols of sulphuric acid are far more potent than sulphur dioxide in affecting pulmonary resistance.

In terms of possible carcinogenic effects it has been demonstrated that, although SO_2 itself is not a carcinogen, when combined with BaP it produces squamous cell carcinoma of the lung in rats. It is not clear whether the "promotion" effect implied by these results is specific for sulphur dioxide. It should also be noted that an identical experiment using hamsters failed to produce tumours. An additive effect of exposure to 5 ppm SO_2 and 5 ppm nitrogen dioxide has been reported by Abe, and a synergistic effect in men exposed to SO_2 and ozone.

In a review of the health effects of atmospheric SO_2, Hickey and co-workers (2) expressed the opinion that the conclusion by some investigators of the harmlessness of exposure to relatively low concentrations of SO_2, as well as sulphites and bisulphites in diets, may be invalid for some individuals. Misleading interpretation of the available data may result from the fact that no account is taken of sulphite oxidase deficient individuals in the human population. Such persons may be unusually vulnerable to mutagenic and other damaging effects of sulphites and bisulphites. The lack of ability to oxidize sulphite efficiently suggests potential health problems particularly in view of the evidence that bisulphite is mutagenic for a number of test organisms. These authors also reviewed previously published data which correlated urban SO_2 concentrations with mortality rates in 45 - 64 year old men. As well, co-consumption for domestic use was found positively correlated with lung cancer death rates for men 45 - 64 years of age for the years 1948 through 1954. In the USA, atmospheric levels of SO_2 for 38 metropolitan populations were found to be significantly and positively correlated with death rates for several cancer and cardiovascular disease categories. The authors concluded that it is imperative that a fallacy of typology relating to potential SO_2 toxicity be recognized and avoided.

In a rebuttal to the above paper, Alarie (2) pointed out that there is no evidence for a wide-spread sulphite oxidase deficiency in humans. He also refuted the conclusions drawn by Hickey as to the carcinogenicity and mutagenicity of sulphite. Alarie pointed out that the group had made several basic misinterpretations of the statistical analysis, as well as misquoting and misreading conclusions from several of the articles and Alarie emphasized the relative safety of SO_2.

Archer and co-workers (3) examined the acute effects and sputum cytology resulting from sulphur dioxide exposure in a smelter population. As was reviewed by these investigators, the acute effects of SO_2 exposure consists primarily of mucous membrane irritation as evidenced by lachrymation, bronchoconstriction, reduced mucous flow and reduced particle clearance from the bronchi. Bronchial constriction, as measured by expiratory flow rates and volumes, has been frequently measured after SO_2

exposure in the range of 5 - 10 ppm (13 - 26 mg/m^3) but is barely detectable at about 1 ppm (2.6 mg/m^3) SO_2. Since these effects are transient they have often been considered to be non-harmful to normal individuals. Pulmonary function tests carried out on 195 smelter workers and 54 control subjects (Table 99) showed that 55 - 65% of smelter workers compared to 45 - 50% of control workers had some decrease in respiratory flow rates during the day, indicating a degree of bronchial constriction. In relation to sputum cytology, there was an increased frequency of moderate and marked atypic samples correlated with an increasing number of cigarettes smoked and an increased number of years of working in the smelter. Cigarette smoking, linked with SO_2 exposure, correlated with a higher lung cancer rate than in control groups. The time-weighted average exposure of the smelter workers to SO_2 was found to range between 0.3 and 4 ppm, with a mean exposure of about 1.4 ppm (3.6 mg/m^3). Small amounts of silicates, sulphates, copper, lead, manganese, zinc and arsenic were also found in the airborne dust.

TABLE 99

Comparison of Sputum Cytology Between Smelter and Control Workers

	Smelter Workers		Control Workers		
Pack-Years of Cigarettes	Normal and Mild Atypia	Moderate and Marked Atypia	Normal and Mild Atypia	Moderate and Marked Atypia	Statistical Significance*
0	53 (65.4)[1]	28 (34.6)	16 (84.2)	3 (15.8)	$P < 0.15$
1 - 15	44 (60.3)	29 (39.7)	8 (53.3)	7 (46.7)	N.S.
16 or more	46 (59.0)	32 (41.0)	14 (63.6)	8 (36.4)	N.S.
Years Worked in Smelter					
0 - 9	83 (63.8)	47 (36.2)	13 (72.2)	5 (27.8)	N.S.
10 - 19	31 (63.3)	18 (36.7)	12 (70.6)	5 (29.4)	N.S.
20 or more	29 (54.7)	24 (45.3)	13 (61.9)	8 (38.1)	N.S.

All nonsmokers vs. all smokers $P < 0.15$
All control workers vs. smelter workers with 20 or more years on job $P < 0.20$

---

* Statistical significance of the difference in percent of moderate plus marked atypia between smelter and control workers

[1] Numbers enclosed in parentheses are percentages

(Source: Reference 3)

Shapiro (232) published an extensive review of the genetic effects of sulphur dioxide, which on hydrolysis reacts to form bisulphite. The principal sources of exposure to exogenous sulphites were considered to be inhalation of sulphur dioxide in the air and ingestion in food, beverages and drugs. It was calculated that at the level of 0.02 ppm, a male doing light work would inhale 2.1 mg over a 24-hour day. An industrial workman, at concentrations of 4 ppm, would inhale 140 mg in an 8-hour day of light work. It was pointed out that an acceptable daily intake of 0.7 mg/kg or 50 mg for a 70 kg person has been established by the FAO/WAO. This author appeared to agree with Hickey et al and noted that sensitive individuals show a response to SO_2 concentrations of 5 ppm with a mild degree of bronchoconstriction measured as an increase in flow resistance. He quoted references in which adverse health effects were detected on occasions when the 24-hour average SO_2 level rose above 0.11 ppm and increased mortality was recorded when it exceeded 0.24 ppm. At the cellular level, bisulphite can be toxic at a concentration as low as 10^{-2} to 10^{-4} molar with inhibition of DNA synthesis, cell division, and mitosis. Chromosome aberrations and mutations have been demonstrated in plants, microorganisms and mammalian cell cultures exposed to similar SO_2 concentrations. No effects have been shown in rats and mice at exposures of 50 - 100 times the human maximum atmospheric exposures based on toxicological studies and mutagenesis tests. On the other hand, epidemiological studies show a clear association between atmospheric SO_2 levels and human respiratory disease. The association between SO_2 exposure and cardiovascular disease and cancer are suggestive, but less firm. Shapiro emphasized that there were two extreme positions in relation to SO_2, one being that it was not harmful, and the other that it was harmful and required careful regulation. The current state of scientific evidence does not allow either of these extreme positions, or a number of intermediary ones, to be determined with a high degree of assurance.

Nobutomo (233) pointed out that it has long been considered that there is a relation between the exposure to heavily polluted air and the increased incidence of lung disorders. This has been demonstrated in animals and in humans and in epidemiological studies, but it is less obvious whether or not prolonged exposure to air pollution levels much below those found under experimental conditions would be an important cause of respiratory disorders.

Nobutomo (233) described the results of sputum cytology studies conducted on 300 - 400 subjects in two areas of Japan, one which was industrialized and considered to have extensive air pollution and a comparable group in a non-industrialized, low environmentally polluted setting. Sputum samples were examined for alveolar phagocytes, neutrophils, eosinophils, lymphocytes and bronchial epithelium. The earliest symptoms of the effect of air pollution was persistent phlegm. However, it was noted that an inflammatory response preceeded the symptoms because the cellular content of the sputum samples collected from those who lived in the polluted area, but had no symptoms, differed from that in samples from equally-symptomless individuals living in less polluted surroundings. It was suggested that the cytological changes in the sputum were probably the most sensitive indicator of the effects of exposure to polluted air and that sputum cytology be included as a screen in assessing the pulmonary effects of air pollution.

In attempts to develop models of chronic bronchitis, many experimental studies have used sulphur dioxide exposure. Vai et al (234) investigated bronchial permeability in the rat, in vivo and in vitro, using tracheal-bronchial explants immediately after SO_2 exposure and 3 months after SO_2 exposure had been discontinued. Exposures were as summarized in Table 100.

TABLE 100

SO_2 Exposure in Four Groups of Rats

Group	SO_2 Concentration (ppm)	Number of Rats	*SO_2 Exposure (h)
1	600 + NaCl mist	5	30
2	600 + NaCl mist	5	60
3	600 + NaCl mist	11	100
4*	600 + NaCl mist	10	100
5 (control)	air	16	

(*) These animals were killed 3 months after SO_2 exposure had been discontinued.

(Source: Reference 234)

No anatomical abnormalities were found in the tracheobronchial samples of the control animals.

Marked anatomical abnormalities in the four SO_2 treated groups were observed, as listed in Table 101. Although many of the lesions have been described by other investigators, the absence of epidermoid metaplasia, mucosal and submucosal edema and epithelial hyperplasic were notable. Differences between groups 1 and 2 were small and the severity of the lesions did not increase when the exposure was prolonged, as in group 3. Optical and ultrastructural aspects of samples obtained from group 4 were normal, and anatomic recovery was achieved.

TABLE 101

Anatomic Abnormalities in Four Groups Of Rats Exposed to SO_2

	Animal Groups			
Lesions	1	2	3	4
Trachea; main and lobar bronchi				
Mucus abnormalities	+	+++	+++	0
Ciliary abnormalities	++	+++	+++	+ or 0
Epidermoid metaplasia	0	0	0	0
Goblet cell hyperplasia	++	++	++	+ or 0
Lysis and desquamation of epithelial cells	+	++	++	0
Intracellular and epithelial edema	+	++	++	0
Mucosal and submucosal edema	0	0	0	0
Inflammatory cellular infiltrates	0	0	0	0
Bronchial vascular abnormalities	0	0	0	0
Glandular hypertrophy	0	+	+	0
Bronchial lymphoid tissue abnormalities	+	+	+	0
Bronchioles and parenchyma				
Epithlial hyperplatia (secretory cells)	0	0	0	0

(Source: Reference 234)

Ferritin uptake and transport were also demonstrated in the explants of each group, with the ferritin uptake and transport pathways for all SO_2 exposed groups reproducing those of the control group.

Most histological damage in mammals relevant to the experimental SO_2 exposure is related to the high water and mucous solubility of the gas. Consequently, the SO_2 lesions are usually proximal, limited and superficial, and vary with exposure. Generally, in both human and animal studies recovery from damage takes place within an hour to a few hours. Only very high exposures of 1,000 ppm for over an hour results in bronchial lesions involving the basal membrane and underlying mucosa. Consequently, it was suggested that SO_2 alone is probably not enough to induce a human-like condition of chronic bronchitis in the rat. For example, in groups 1, 2 and 3, what was described was similar to a proximal, subacute bronchitis. The toxicity of SO_2 was probably increased by the absorption of the gas by aerosol droplets in the sodium chloride mist, and the fact that the concentration of SO_2 was very high, being more than 1000-fold greater than the levels in urban air.

When groups 1, 2 and 3 are compared, it would seem that prolonged SO_2 exposure does not increase anatomical damage. This finding supported the belief that for an intermittent SO_2 exposure of 600 ppm 2 to 5 hours per day, the bronchial mucosa could reach a steady-state between injury and repair. This is more believable when it is considered that: 1) regeneration of mucous membrane starts one to two hours after trauma, whether mild or severe; 2) peak multiplication of cells adjacent to a desquamated area is probably within 24 hours of trauma, and 3) cell damage varies considerably throughout and along the bronchial mucosa, allowing cells from the undamaged or newly-repaired area to migrate into the injured areas.

A study by Wilhelms (referred to by Vai et al, Ref. 234) showed that the anatomical event leading to complete regeneration of mucosa in rat trachea lasted about 6 weeks, which agreed with the findings in the rats from group 4 in the Vai study.

In the Vai investigations, quantitative data was not obtained, but known amounts of ferritin were dropped on the explant. Ultrastructural studies using the electron microscope of rapidly fixed samples showed that differences, if any, were negligible. No difference in the uptake and transport of ferritin along the bronchial tree was noted, which was unexpected when considering the difference of SO_2 uptake and subsequent damage occurring between the nasal pharynx and distal airways. On the other hand, comparison of group 1 with groups 2 or 3 suggested that prolonged SO_2 exposure increased the permeability to ferritin and that SO_2 related epithelial damage was similar.

A further comparison of the control group with the SO_2 exposed animals showed that the selectivity of the bronchial lymphoid and epithelial lining tissue for the uptake and transport of ferritin disappeared. As early as five minutes after the ferritin was injected in vivo or dropped on the explants, the tracer accumulated under the basal membrane in the SO_2 exposed animals and disappeared from the epithelium.

The major finding in this study was the persistence of an abnormal permeability up to 3 months after stopping exposure to SO_2. This could lead to the penetration of the tracheobronchial mucosa and submucosa by macromolecules. Whether the long-term effects of bronchial irritation induces a particular bronchial reactivity, as upper respiratory tract infections do in normal subjects, is possible, but not known.

The studies quoted have indicated the quality of the pollutant SO_2 and have established reasonable levels that can be permitted in the mine atmosphere. In 1978 the authors commented that the latest studies indicating the spectre of synergism with other contaminants suggested that the "reasonable threshold limit value" for SO_2 should be lowered from 5 ppm to 3 ppm. In fact, the ACGIH-TLV is 2 ppm or 5 mg/m^3.

Other factors requiring consideration are humidity and dust levels.

Only the most limited and meagre information is available from recent research regarding the possibility that SO_2 is implicated in the development of neoplasia. Much more study is required in this area before conclusions can be drawn, and revision of SO_2 levels may be in order.

When all factors are taken into consideration, and the possibilities of synergism and neoplasia are contemplated, a major priority in the near future should be the development of sulphur-free diesel fuel.

4.5 Diesel Particulate

In the 1978 edition of this document, the authors expressed concern with the potential toxicity of diesel particulate, both because of its inherent propensity to affect pulmonary function and because of its unique facility to act as a carrier of other pollutants, such as acid, SO_2, PNA's, etc. At that time, experimental data delineating the toxic potential of diesel particulate was relatively meagre and emphasis was to the rather poorly defined association between PNA's known to be present in exhaust and the respirable particulate or soot. Thus the bulk of the discussion was carried in Section 4.6 titled "Polynuclear Aromatic Hydrocarbons".

Since that time, a plethora of studies have been brought to varying extents of completeness and have been reported in the literature and at scientific meetings. As was reviewed in Section 3.4.7, much has been added to our knowledge of the chemical nature of the PNA content of diesel exhaust and its particulate. Of even more significance is the toxicological information which has been generated since release of the original review (1). Because of the emphasis placed on the particulate component of exhaust, the present authors have shifted their main discussion to section 4.5, Diesel Particulate. The extent of the information flow which has been forthcoming in this area is further emphasized by the necessity to present subsections dealing with deposition, mutagenicity and toxicology of diesel particulate and its attendent diesel components.

Because much of the information which was available at the time of writing of the 1978 document has been superceded by new data of a much more relevant nature, a large part of Sections 4.5 and 4.6, as presented in 1978, have been entirely omitted in the present version of the publication.

Above ground diesel vehicles have been estimated to contribute 2,060 tons per year of particulate matter to the U.S. environment (235).

Generally, the particles under consideration are less than 100 microns in diameter and are exclusive of those substances with a specific toxic effect, such as silica.

The particles which are able to penetrate to the alveoli (usually less than 5 u in diameter) are considered respirable dust, but it is emphasized that the remaining particles from an aerosol or dust cloud are also important as possible causes of bronchitis when deposited in the upper portions of the respiratory system (236).

Particulates greater than 2 microns which are inhaled are impacted on the nasal passages. Particles 2 u to 0.05 u deposit by gravitational sedimentation at the bronchial-bronchiolar levels in the lung. Particles ranging in size from 0.01 u to 0.05 u are particularly likely to settle in the alveoli (237, 238).

Suspended dust is subjected to the same forces as other falling objects in that the action of gravity pulls the particle down and the resistance created by dynamic drag tends to keep the particle afloat. In the case of extremely fine particles of less than 1 micron, Brownian movement reduces the tendency to settle. Fine particles are really an integral part of the air mass enclosing them and they will behave as the air mass does (239).

Particle shape, as well as density, determines the effective aerodynamic size of irregularly-shaped particles. Mineral particles such as quartz and coal are aerodynamically equivalent to spherical particles 1/2 to 3/4 of their directly-measured diameters (240).

The deposition of a particle in the pulmonary system depends on the aerodynamic properties of the particle, the dimensions of the airways and the person's breathing pattern. Definitions of aerosols, gravitation, sedimentation, inertial impaction, diffusion, and the effect of particle shape on aggregation, as well as the pattern of breathing on particle deposition are all significant (241, 242, 243, 244, 245).

The dust that settles on the mucous film of the dead-space epithelium is conveyed to the pharynx on the mucous ciliary blanket within three to four hours. As described in Section 3.1, if there is overproduction of mucous such as in acute and chronic bronchitis, the mucous blanket becomes thicker than 5 to 25 microns and the dust fails to move on the beating cilia and particles are not moved up and out of the lungs and airways until thinning of the mucous and coughing take place. However, it should be noted that the epithelium of the airways may be protected for brief periods from toxic particles and alpha particle radiation when mucous is overproduced, as in the case of the smoking miner with chronic bronchitis (244).

The tracheobronchial system plays the primary role in the clearance of the lungs. Particles on the mucociliary escalator are found in the mucous blanket from the terminal bronchioles to the pharynx. A particle impacts on a thin layer of mucous which covers the epithelium, and is continually swept up by ciliary action at the rate of around 3 mm per minute. The ciliary transport is influenced by ionic charges, the presence of oxidants, cigarette smoke, high concentrations of oxygen and low concentrations of adenosine triphosphate (244).

The alveolar transport system is the next step of the clearance mechanism. Within 24 hours, about 50% of the material deposited is cleared by the surface transport system from the proximal areas of the lobule along the surface of the respiratory bronchi. A second slower process has a clearance time of close to 100 hours and probably involves septal transportive material that has been phagocytozed. Thirdly, a mechanism with a 50% clearance of around 50 to 100 days, involves foreign particles that have reached dead-end pathways in perivascular and peribronchial spaces. With a large amount of particulate matter, the cellular transport mechanism may be overwhelmed. If this occurs many particles can be seen lying free in the alveolar region along with alveolar macrophages (244).

Davies (243) reported that during steady breathing of an aerosol, deposition in the lung of 0.5 um diameter particles varies with the degree of expansion of the lungs, being greater when the expiratory reserve volume is less. This is due mainly to the decreased thickness of the sheath of reserve air which encloses the tidal air in the alveolated airways.

Calculations based on lung anatomy and particle mobility indicate that a given particle which is transferred to the reserve air after inhalation will remain airborne in the reserve and residual air for up to nine breaths before it is deposited on the alveolar wall.

Further examination reveals that particles remain airborne in the lungs for at least five breaths. Washout experiments have proved them to still be present in the reserve air after nine breaths.

During the steady breathing of an aerosol, all parts of the lung acquire a representative concentration of particles. This is not so when only a single inhalation of aerosol is taken (243).

After being engulfed by alveolar macrophages particles are eventually excreted on the same mucous escalator which removes the material trapped in the upper airways. Some dusts may be retained permanently within the lung or hilar lymphatics and may cause tissue damage. Therefore, the effect of inhaled dust depends on the region of the lung where it is deposited, as well as the toxicity of the inhaled particle (236).

Small particles penetrate further into the lungs and are therefore cleared more slowly than large particles. The rate of clearance by the mucosa is not in itself sensitive to the particle size nor to the number of particles deposited, unless the particles have some specific toxic effect. The clearance of the mucosa appears to be essentially complete in less than 24 hours. The great majority of all particles trapped on the mucosa during a working shift are, in fact, cleared within a few hours and do not accumulate during regular daily work. The ciliary lining of the nose is also cleared within hours (236).

Dust penetrating to the alveolus is engulfed by the alveolar macrophages within a few hours after deposition. There is some uncertainty about the amount of dust which is cleared from the lung during daily exposure by industrial workers.

Using published functional deposition values, and assuming a coal miner working in poor conditions, it is calculated that a miner could receive a total lifetime alveolar dust load of 1,000 grams. However, to find more than 40 g of dust in the lungs of miners on post-mortem examination is considered unusual (236). On the other hand, calculations by Policard from studies in French coal miners indicated that pulmonary clearance was 98 - 99% effective (see Section 3.1).

Another researcher calculated that a coal miner working the normal number of hours inhales between 100 and 150 g of dust in a year. Of the dust inhaled, about 1 to 10 g was deposited in the alveoli, but only approximately 0.5 g was permanently retained. It should be noted that pulmonary deposition of 5 g of silica would be fatal (244). This suggests that alveolar clearance mechanisms are highly efficient, or the amount of deposition is not as great as thought (236).

Of some interest is the statement that dust does not appear to accumulate in the lungs of miners at a steady rate, and the amount of dust in the lungs at any given time is not simply a function of the total number of days or years of exposure. Rather, the dust tends to rise rapidly during the first 10 years or so, and then levels off or increases much more slowly.

Caution is recommended in assessing laboratory-derived data as industrial dusts may not be cleared from the lung at the same rate as experimental aerosols produced in the laboratory. Although cigarette smoking may be associated with some change in the aerosol deposition pattern and in the rate of mucociliary clearance, smoking is likely to be of minor importance in comparison with its known association with chronic bronchitis and its attendant pulmonary disability (236).

A simple mathematical model to describe the influence of chronic inhalation of dust on lung retention has been proposed. The validity of the theoretical model was assessed using animal studies and information obtained on lung content measurements of miners (246, 247).

Deposits in the alveolar region are removed by the alveolar clearance at a very slow rate onto the bronchial epithelium by means of alveolar phagocytosis. Depending on the chemical nature, fractions of the deposited dust succeed in penetrating the alveolar walls and enter the lung tissue, where it is stored in malignant dust cell granuloma or are partially removed by lymphatic clearance. The amount of dust remaining in granuloma is defined as tissue retention which, in the case of diminishing lymphatic clearance, is essentially permanent.

The formula recommended as describing particulate retention was given as being:

$$R_L = \frac{dD_A}{dt} \frac{1 - e^{-Kt}}{K} \cdot$$

D_A = alveolar deposition
K = clearance rate factor

$$R_L = D_A - C_A - C_T \text{ with}$$

C_A = alveolar clearance
C_T = lymphatic clearance
R_L = lung retention

The equation indicates that, in cases of chronic inhalation, the lung retention will not exceed a certain level unless the elimination mechanisms break down (excessive deposition rate) or a substantial portion of the dust infiltrates the lung tissue which would be revealed by pronounced occurrence of the lymphatic clearance. Furthermore, a retention maximum would be proportional to the deposition rate and inversely proportional to the clearance rate factor.

Human and animal data suggested that the equation was suitable for at least a semi-quantative description of normal lung retention during chronic inhalation. The human work indicated that normal lung retention, which is predominantly alveolar, was not proportional to the time of chronic exposure, but reached a maximum value depending upon deposition rate and therefore upon an ambient concentration of respirable dust (236).

Review of the "V" or "AQI" formula in the original edition of this document makes it clear that French and Mildon consider the presence of respirable combustible dust (RCD) in diesel exhaust to be the most significant health hazard pollutant. The RCD factor appeared in the AQI equation three times and, because of the suggestion of a synergistic interaction with SO_2 and NO_2, the presence of RCD has the greatest impact on the value derived from the calculation.

The rationale for placing such a heavy emphasis on the RCD resulted from the knowledge that diesel soot is a respirable particulate composed primarily of carbon black to which toxic and carcinogenic components of diesel exhaust could attach and be carried deep into the lungs.

The potential significance of diesel particulate as a primary health hazard of diesel exhaust is obviously in agreement with others judging from the comments made by various investigators at the EPA Diesel Emission Symposium held in North Carolina in October 1981 (4), thus the heavy emphasis placed on this component in the Health Effects Index formula appear to be justified.

As a result of the concern for the potential hazards of diesel particulate there has been an increased impetus and activity in chemically characterizing (see section 3.4.7) and toxicologically defining diesel particulate. The review of the available data, presented below, confirms the authors' concerns for the potential health effects of diesel particulate, while at the same time modifying their interpretation of its relative importance as a health hazard. Research into diesel particulate has taken several avenues, including the study of the physical deposition of particles, the mutagenicity of extracts using in vitro bacterial test systems, human and animal cell-line mutagenicity testing and toxicological investigations of the effects of inhalation exposure to diesel exhaust, intratracheal instillation and skin application of diesel exhaust condensates or extracts. The literature will be reviewed under appropriate headings in order to assist in delineating current knowledge in each of these areas.

It is suggested that this section be reviewed in conjunction with Sections 3.4.7 and 3.4.8, in which various aspects of diesel particulate and PAH's are reviewed. An excellent review of all aspects of diesel particulate can be found in the Proceedings of the EPA Diesel Emission Symposium held in Raleigh, North Carolina, October, 1981 (4).

Particulate exposures in underground mines have long been recognized for their etiology in occupational disease. Silica (quartz), coal, and numerous metal and nonmetal ores have contributed to both acute and chronic illness.

As discussed above, physical properties such as size, shape and density are important to pulmonary deposition and retention which varies considerably, depending primarily on the amount of energy exercised against the contributing material. For example, slushing, crushing, and mucking generally produce relatively large particles, while blasting or drilling generate a much smaller particle size distribution.

Particulates arising from diesel emissions are of special concern from three aspects: 1) most diesel particulate is in the respirable range, 2) its physical characteristics are such that NO_2, SO_2, acids, and other toxic agents may be adsorbed and, thus, carried into the deeper areas of the lung, 3) it adsorbs polynuclear aromatic hydrocarbons (PNAs), some of which are established animal carcinogens.

Diesel particulate composition varies widely according to engine speed, load, and to a lesser extent, to the type of engine and fuel used. Straight chain saturated hydrocarbons, aldehydes, ketones, esters and glycidols, aromatic ethers, cyclic ketones and hydrides, alkylbenzenes, phenols, dihydric phenols, hydroaromatic and polynuclear aromatics have all been identified in exhaust.

It is clear that the complexity of diesel exhaust is incredible and its chemical and toxicological delineation would boggle the mind of the most ardent scientist.

The toxic effects of exposure in underground mines to silica, coal and ore dusts are well documented, but are not specifically related to diesel and explosives use and, thus, will not be reviewed here except as they may relate to the possible enhancement of the toxic effects of other mine air contaminants.

The toxicity of inhaled particles is complex and depends on the chemical and physical nature of the particles, size distribution in an aerosol, and on the efficiency with which it is removed from the respiratory tree. The presence of heavy dust, which may be encountered in the mining environment, can adversely affect the lung clearance mechanisms which result in less efficient removal of particulate PNA's, and thus possibly increase the carcinogenic risk. Enhancement of tumourogenicity of benzo(a)-pyrene has been demonstrated when the chemical was adsorbed to carbon particles as compared to the chemical alone, presumably as a result of its delayed clearance from the lung.

Several studies have implicated the role of particles in facilitating the transport of PNA's into cell membranes thus increasing their availability for intracellular microsomal activation. Cellular availability of PNA's is enhanced by the associated particulate being phagocytozed by macrophages, by facilitating uptake by cell membranes or by puncture of the cell membrane. For example, Lakowicz et al (271) demonstrated that chrysene enters phospholipid bilayers much more rapidly from a silica-adsorbed state than from a microcrystalline state.

The ACGIH, in Appendix A of the Threshold Limit Values (248), lists those substances in industrial use that have proven to be carcinogenic in man or have induced cancer in animals under appropriate experimental conditions. Two categories are included: those which the ACGIH consider not to have been sufficiently evaluated to permit assigning a TLV, and those for which a TLV has been assigned. The latter list includes particulate polycyclic aromatic hydrocarbons (coal tar pitch volatiles), as benzene solubles, with a TLV of 0.2 mg/m^3.

The comparable MSHA and OSHA standards are listed as the benzene soluble fraction of "coal tar pitch volatiles" which includes the fused polycyclic hydrocarbons that volatilize from the distillation residues of coal, petroleum, wood, and other organic matter. Specifically mentioned as examples are anthracene, BaP, phenanthrene, acridine, chrysene, and pyrene. The limit for both groups is also 0.2 mg/m^3 and was adopted from the ACGIH 1968 and 1973 Threshold Limit Values.

The current ACGIH TLV and MSHA and OSHA standards for carbon black are 3.5 mg/m^3 on a TWA basis. NIOSH recommended, in addition to the 3.5 mg/m^3 TWA, that the limit should be 0.1 mg/m^3 TWA in the presence of polycyclic aromatic hydrocarbons.

In view of the expected increase in the use of diesel engines in cars and light-medium duty trucks in the next 5 years, General Motors instituted an extensive research program related to the potential health effects of diesel exhaust, especially particulate and its associated toxicants such as PAH's. Other groups, such as the EPA, Southwest Research Institute, Batelle-Columbus Laboratories, Lovelace Biomedical and Environmental Research Institute, and various University groups, are engaged in related research programs.

Preliminary findings from many of these groups have been made available at meetings such as those sponsored by the EPA, CRC, etc., and in in-house publications. These reports, together with studies published in the open literature, are included in the following sections and should provide a timely summary of the current state of knowledge relating to the potential health effects of diesel particulate.

Vostal (494), in a recent issue of the Bulletin of the New York Academy of Medicine provided an excellent summary of the General Motors research activities. An additional reference with comprehensive description and evaluation of the results of several groups' efforts in this area can be found in the recently-published Proceedings of the EPA 1981 Diesel Emission Symposium (4).

4.5.1 Deposition and Metabolism of Diesel Particulate

A number of theoretical and experimental studies have demonstrated that inhaled particles are deposited in different regions of the respiratory tract, depending on their effective aerodynamic size. Particles that are deposited in the nasopharyngeal and tracheobronchial regions of the respiratory tract do not remain there long. Such particles are normally removed to the pharynx, often by cilia action and swallowing within a matter of hours. Consequently, extraction of toxic species from these particles takes place predominantly in the stomach where their residence time is short.

Exceptions to this rule occur in the case of particulate whose constituents are either extremely soluble or paralyze cilial action. On the other hand, particles deposited in the pulmonary region may remain there for weeks or even years in intimate contact with approximately 30 square meters of microscopically-thin alveolar membranes which separate the bloodstream from inhaled air.

Residence time is probably not the only factor controlling extraction efficiency, since even comparatively insoluble species are "desorbed" from particulate at a surprising rate in the pulmonary region. At least part of the extraction phenomenon is probably due to particle size, with the rate of extraction per unit mass depending upon the surface-to-volume ratio of a particle. Chemical effects and membrane transport characteristics may also play a determining role (249).

A model of the pulmonary anatomy defines two clearance phases from the pulmonary region, one from the compartment consisting of the respiratory bronchioles and all the structures connected with the alveoli and the other includes phases that depend on macrophages coupled to the ciliary-mucous transport process.

Early attempts to define particulate deposition emphasized carbon (nutshell charcoal) or Teflon particles coated with BaP or metal particles.

In the charcoal-BaP studies (250), two particle size ranges of 0.5 -1.0 u and 15 - 30 u were used with the proportion of BaP:carbon being about 1:1. The pulmonary retention of the carbon particles was measured by tagging the particles with ruthenium-103. ^{103}Ru-labelled carbon particles and BaP-coated carbon were mixed together and injected intratracheally into mice. The amount of radioactivity delivered to each mouse was determined by means of a whole-body counter and was used to calculate the amount of carbon particles as well as BaP administered.

Analysis of the BaP content of mouse lungs was done by chemical extraction and subsequent spectrofluorometric analysis. It was found that BaP crystals administered intratracheally without carbon were eliminated rapidly with 50% of the initial dose being removed within 1.5 hours and greater than 95% within 24 hours.

However, BaP attached to carbon particles disappeared slowly from the lungs. Fifty percent of the initial dose was cleared in approximately 3 hours, while at 4 days, approximately 40% of the dose still remained.

It was concluded that the physical adsorption of BaP to physiologically "inert" carbon particles increased the lung's retention of BaP more than 20-fold over that for unadsorbed BaP. The pulmonary clearance of carbon particles alone was even smaller. After 7 days, 50% of the particles had not been cleared. This data was interpreted as indicating that BaP was being eluted from the carbon, at a rate of approximately 45% per day. Therefore, at least 15% of the initial BaP dose was available to react with respiratory epithelial tissue during the first 4 days after exposure. These experiments were all carried out using carbon with a particle diameter of 0.5 to 1 um (250).

When 15 - 30 um carbon particles were used, the disappearance of BaP was even slower, with 50% being eliminated within 4 - 5 days. However, this rate was approximately the same as for carbon particles themselves, indicating that little BaP dissociated from the large carbon particles during the time of retention by the mouse lung (250).

In another study, 5 micron Teflon particles were coated with various metal particles including lead, carbon and uranium (Table 102). After remaining 7 days in a water bath at 37°C, greater than 50 percent of the coat remained. Even after one month in the water bath, coatings greater than 50 percent were retained except in the case of lead and uranium (251).

Aerosols of coated particles were produced and about 90% of the particles occurred in single form. The aerodynamic diameters were examined, using lead as an example. Evaporation of 100 mg of lead gave a mean thickness of the coating of 0.035 micron. This was an increase in aerodynamic diameter of 0.3 micron, which was no more than the differences in the mean diameter of non-coated particles. The aerodynamic diameter of 5-micron, non-coated Teflon particles was 7 microns. The increase in the aerodynamic diameter for a coat of the same thickness was less for the other elements studied since they all had lower specific densities (251).

TABLE 102

Percentage of Particles Coated*

	A			B			C		
Element	>50%	<50% >0%	>0%	>50%	<50% >0%	>0%	>50%	<50% >0%	>0%
Aluminum	100.0	0	0	99.6	0.4	0	94.2	4.3	1.5
Beryllium	98.7	1.3	0	86.0	14.0	0	71.9	25.7	2.4
Carbon	100.0	0	0	97.1	2.9	0	92.5	7.0	0.5
Chromium	97.5	0.7	1.8	96.3	0	3.7	88.9	0	11.1
Lead	95.2	3.5	1.3	55.2	38.6	6.2	47.1	33.3	19.5
Manganese	79.0	17.6	3.4	77.1	19.6	3.3	70.0	22.7	7.3
Silver	100.0	0	0	100.0	0	0	98.9	1.1	0
Uranium	100.0	0	0	87.6	12.4	0	0	40.9	59.1

(*) A, immediately after dispersion in saline solution and ultrasonic treatment; B, after one week; and C, after one month in saline solution at 37°C.

(Source: Reference 251)

Phagocytosis in vitro by alveolar macrophages during 1.5 hours of exposure to the particles coated with lead, silver and carbon was studied by Camner and his associates (251). Particles coated with carbon were phagocytozed to a higher degree than the particles coated with lead and silver, as can be seen in Table 103.

TABLE 103

Particles Per Macrophage After 1.5 Hours

Rabbit	Carbon-Coated Particles	Lead-Coated Particles	Silver-Coated Particles
1	1.08	0.55	0.72
2	1.29	0.99	0.96
3	0.76	0.65	0.43
4	0.83	0.41	0.33
5	1.12	0.46	0.75
6	0.97	0.59	0.58
Mean	1.01	0.61	0.63

(Source: Reference 251)

The higher rate of carbon particles being phagocytozed has been borne out by other studies (250).

The important aspect for consideration is that the adsorbed coat found on particulate matter is of biological significance (251).

Teflon particles smaller than 4 microns are difficult to produce, but even 5 micron particles with an aerodynamic diameter of about 7 microns may be deposited in the bronchi, bronchioles and to a considerable extent in the peripheral alveoli. In an experiment with 6-micron polystyrene particles, about 60 percent remained in the rabbit lung 24 hours after inhalation (252). In experiments with human subjects using 6-micron Teflon particles as the inhaled aerosol about 20 percent of the particles remained in the lungs after twelve hours (251, 253).

Ferin (254) studied rats exposed to TiO_2 particles in a dusting chamber. Five minutes after beginning the exposure, few particles were seen in the alveoli, so that one could make good judgement about their disposal. Following a 30-minute inhalation, the particles were numerous; they were located not only on the alveolar walls, but also intracellularly. Some of the alveolar macrophages laden with particles had, at that time, already been carried onto the "ciliary escalator". Two days after commencing the 7-hour exposure, the number of alveolar macrophages was substantially increased and most particles were found intracellularly.

The retention half-time in these experiments with a single TiO_2 exposure was found to be 14 days for the first phase, and 88 days in the second phase. It is generally believed that the overwhelming accumulation of dust in the lungs which leads to lung pathology must be blamed, at least partially, on failure of the clearance mechanisms (254).

The inhalation of coal, even in small quantities, leads to a decrease of alveolar clearance in rats. After heavy doses of dust, the rate of alveolar clearance becomes negligibly small; moreover, the clearance function does not recover appreciably. Inhaled quartz decreases the rate of lung clearance in about the same way, but the clearance activity partly recovers (255).

Anesthetized rats or hamsters were given intratracheal injections of particles suspended in saline and subsequently dispersed ultrasonically. Four hours, one day or 3 days following the injection, the animals were sacrificed. The lungs were washed and the volume of free cells in each saline wash was 5 times greater than the tissue volume. In general, the increase in free cells (macrophages) was maximal one day after the injection, but remained elevated from control levels at 4 hours and 3 days. Coal dust, carbon, chrysotile, iron oxide and barium sulphate were all associated with an increase in the recovery of free cells. When the particle size of the coal dust was reduced while the total mass was kept constant, the response was enhanced (256).

Soderholm, of the GM Biomedical Laboratories (257), developed a compartmental model to explain the deposition, transport and clearance of insoluble sub-micron particles from the pulmonary system (Figure 100). The major difference between this model and earlier models, such as the ICRP clearance model, is that it describes a wide range of experimental inhalation conditions and provides compartments and parameters that apply even to expcsure at nearly maximum tolerated doses. The application of the model has so far shown that no single set of model parameters fit all the data available for all experimental approaches such as the two main ones of exposing animals for short periods of time followed by measurement of change in lung burdens, or the second approach of exposing the animals for a long period of time with periodic measurement of lung burdens. The author suggested that the differences may be related to deficiencies in the model or to actual differences in the response of the respiratory system to particulate during different exposure situations. The amount of particulate in the lungs of chronically-exposed animals suggests a linear build-up over time and indicates that after excessive exposure, some portion of the deposited particulates may be retained in the lung for relatively long periods.

Wolff and co-workers (258, 259, 466) measured the deposition of 0.02 and 0.1 um $^{67}Ga_2O_3$ particles in Beagle dogs using external detection, as well as the deposition and retention of 0.1 um aggregated

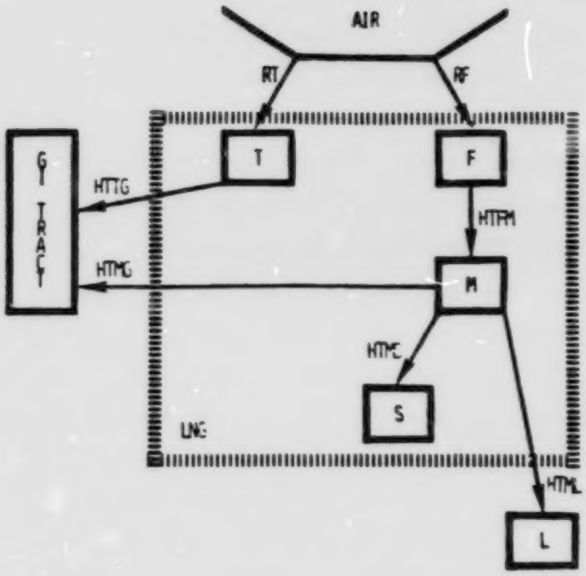


FIGURE 100

Compartments and parameters in the model

Compartments;

T = 'tracheo-bronchial'
F = 'free particulate' on deep lung surfaces
M = 'macrophages' and other scavenger cells
S = 'sequestered particulate'
L = 'lymph nodes' draining lung
G = 'GI tract'
LNG = 'total lung

Parameters:

RT = deposition rate into T
RT = SDT * C
SDT = specific deposition rate into T
SDT = DET * VM
DET = deposition efficiency of compartment T
C = concentration of airborne particulate
HTxy = clearance half-time from compartment 'x' to compartment 'y'
RF = deposition rate into F
RF = SDF * C
SDF = specific deposition rate into F
SDF = DEF * C
DEF = deposition efficiency of compartment F
VM = minute volume

(Source: Reference 257)

aerosols in rats to simulated diesel exhaust aerosols. Dogs were exposed, nose only, to the test aerosol for one half hour and after exposure whole body radioactivity was determined. Fractional deposition in the nasal, pharyngeal and thoracic regions was measured using computerized graphic display and analysis. Rats were exposed whole body in a multi-tiered chamber housing all 144 animals. Seventy-two rats were exposed for five hours and 72 for 5 hours per day for 3 days. In each group, 42 rats were sacrificed immediately after exposure ended and then 6 rats at 1, 2, 4, 8 and 16 days after exposure for tissue counting. Whole body counting was performed at the same time intervals. The average total deposition of 0.02 um aerosols in dogs was 53 ± 24%, which was higher than for the 0.1 um aerosols at 39 ± 9%. The regional deposition of the inhaled aggregate aerosols is presented in Table 104.

TABLE 104

Mean Total and Regional Deposition of Inhaled Ultrafine Aggregate Aerosols in Dogs

Compartment	0.1 um	0.02 um
Nasopharyngeal	7%	9%
Tracheobronchial	7	12
Pulmonary	25	32
TOTAL	39%	53%

(Source: Reference 258)

Figure 101 shows the short-term retention of the irregularly-shaped aggregate particles in the thorax. The authors concluded that the deposition values were in good agreement with data from previous studies with larger sized particles (Figure 102).

In a more recent report, Wolff et al (467) conducted whole-body exposures of $^{67}Ga_2O_3$ aggregate aerosols in rats. One group of animals was exposed for 5 hours on one occasion and a second group

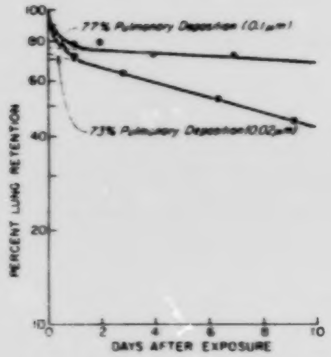


FIGURE 101

Short-term retention of the irregularly shaped aggregate particles in the thorax as determined by gamma camera analysis. The lines through the data are best fit two-component exponential function.

(Source: Reference 466)

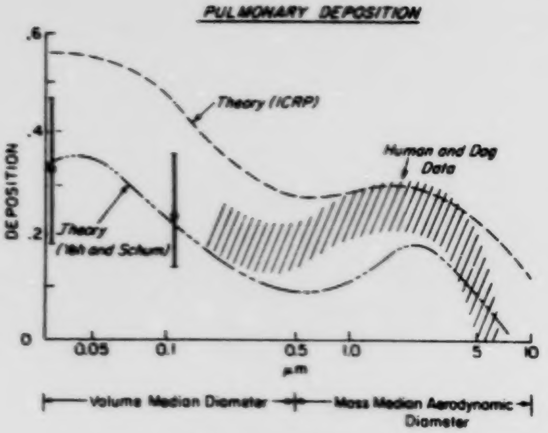


FIGURE 102

Comparison of mean pulmonary deposition (± SD) of 0.1 and 0.2 um irregularly shaped aerosols (●) with that of spherical aerosols. The mid range of deposition data (shaded) taken from human experiments (Lippmann, 1977) and dog experiments (Cuddihy et al, 1973) is shown. Monodisperse and polydisperse aerosols were used in the human and dog exposures respectively. Also shown are theoretical predictions for deposition in humans. The ICRP (Morrow et al, 1966, Fig 12) curve (—) was for deposition of polydisperse aerosols with 750 ml tidal volume. The curve (-· ·-) of Yeh and Schum (1980) was for monodisperse particle aerosols with 750 ml tidal volume and 15 breaths per minute.

(Source: Reference 466)

for 5 hours a day on three consecutive days. Rats were sacrificed at various times following exposure and lung burdens determined up to 15 days after exposure. The whole-body exposure data was then compared to the distribution of the same aerosol aggregates in rats exposed by nose-only inhalation. Tables 105 and 106 summarize the tissue distribution of the radioactive particulate under both whole-body and nose-only exposure conditions. As can be seen, the results are quite similar between the two types of exposures. The values represented a deposition of about 15% of the inhaled particles which is similar to values obtained for nose-only exposures. Radioactivity in the GI tract was about 1.6-fold greater for whole-body exposure than for nose-only exposure, resulting mainly from ingestion by grooming of the pelt. About 60% of the pelt burden was calculated to be ingested following whole-body exposures (Table 107).

TABLE 105

Tissue Distribution of Activity[A] as a Percentage of Initial Body Burden at Various Times Post 1 Day Whole Body Exposure

Tissue	1/2 hr	1 day	3 days	6 days	9 days	15 days
	a)	a)	a)	a)	a)	a)
Lung	22[A] ± 7	18 ± 5	17 ± 2	13 ± 3	14 ± 2	12 ± 3
G.I.	26 ± 19	21 ± 10	4 ± 2	2 ± 1	1 ± 0.2	1 ± 0.5
Pelt	46 ± 22	23 ± 11	12 ± 4	8 ± 4	4 ± 2	6 ± 2
Nares	0.7 ± 0.3	0.02 ± 0.02	0.01 ± 0.01	0.003 ± 0.003	0.003 ± 0.002	0.005 ± 0.005
Liver	[B]	[B]	0.02 ± 0.01	0.5 ± 0.3	0.5 ± 0.3	0.3 ± 0.1
Carcass	2 ± 2	3 ± 2	2 ± 1.5	1 ± 0.3	1 ± 0.5	1 ± 0.6

[A] Mean ± S.D. of values from 3 males and 6 females.

[B] Not available.

(Source: Reference 467)

TABLE 106

Tissue Distribution of Activity as a Percentage of Initial Body Burden at Various Times Post 1/2 Hr Nose-Only Exposure

Tissue	1/2 hr	1 day	3 days	6 days	9 days	15 days
	a)	a)	a)	a)	a)	a)
Lung	37[A] ± 4	39 ± 8	29 ± 7	25 ± 4	23 ± 3	22 ± 2
G.I.	22 ± 4	13 ± 8	7 ± 4	2 ± 1	1 ± 0.3	0.5 ± 0.3
Headskin	26 ± 3	9 ± 3	7 ± 5	1 ± 0.6	1 ± 0.5	0.5 ± 0.3
Pelt	7 ± 3	6 ± 4	3 ± 1.4	3 ± 2	1 ± 0.3	1 ± 0.8
Nares	0.7 ± 0.3	0.3 ± 0.3	0.1 ± 0.05	0.04 ± 0.02	0.03 ± 0.03	0.02 ± 0.01
Liver	0.01 ± 0.008	0.02 ± 0.02	0.2 ± 0.1	0.4 ± 0.2	0.3 ± 0.2	0.2 ± 0.1
Carcass	2 ± 1	2 ± 0.7	2 ± 1.7	1 ± 1	1 ± 1	1 ± 0.7

[A] Mean ± S.D. of values from 3 males and 3 females.

(Source: Reference 467)

TABLE 107

Lung Burdens Following Exposure to $^{67}Ga_2O_3$ Aerosols

	A. Whole Body Exposures				B. Nose-Only Exposures	
	Group 1		Group 2			
	Male	Female	Male	Female	Male	Female
Average Air Concentration (μCi/L)	0.0130	0.0130	0.0121	0.0121	1.6	1.6
Absolute Lung Burden (μCi)	0.18 ± 0.04	0.14 ± 0.02[A]	0.49 ± 0.10	0.40 ± 0.09[A]	2.4 ± 1.1	1.7 ± 0.9
Normalized Lung Burden [(μCi/hr)/(μCi/L)]	2.8 ± 0.6	2.1 ± 0.3[A]	2.7 ± 0.6	2.2 ± 0.4[A]	3.0 ± 1.4	2.1 ± 1.1
Body Weight (kgm)	0.309 ± 0.021	0.195 ± 0.013[A]	0.313 ± 0.022	0.194 ± 0.012[A]	0.236 ± 0.014	0.158 ± 0.015
Lung Burden/Body Weight [μCi/hr)/(μCi/L)/kgm]	9.0 ± 1.9	10.7 ± 1.5[A]	8.6 ± 1.9	11.3 ± 2.0[A]	12.6 ± 5.8	13.3 ± 7.1
Percent Deposition[B]	15	16	14	17	16	16

[A] Significantly different than corresponding values for males ($P < .05$).

[B] Based on assumed minute volumes of 310 mL for males and 230 mL for females (Mauderly and Kritchevsky, 1979).

(Source: Reference 467)

The data showed that repeated exposure at low levels was the same as the summation of independent treatment events. Summing the one-day results to predict the three-day results were in good agreement with the observed retention curve. About 40% of the initial lung activity was cleared by 15 days, and suggests that the relatively insoluble aerosols of $^{67}Ga_2O_3$ are retained in the lung for long periods of time.

In a recent publication (468) the Lovelace Biomedical Group reported on further quantitative evaluations of diesel soot particulate clearance from rat lung following sub-chronic exposures. Rats were exposed to diesel exhaust particulate generated by an Oldsmobile 5.7 Litre diesel engine, running on a 7-mode urban cycle to diesel soot concentrations of 4100, 940 and 150 mcg/m^3. Exposure was for 7 hours a day, 5 days a week for an 18-week period. Diesel soot clearance from the lungs following the sub-chronic exposure was quantitatively determined by isolation of the soot contained in the lungs with measurements using spectrophotometric techniques at 1 day and 1, 5, 15, 33 and 52 weeks after the termination of exposure. A group of control animals was carried along with the exposed groups and the lung burden of particulate in the control animals was subtracted from that of the exposed rats to obtain a net lung burden expressed as micrograms of soot per gram wet lung tissue. Figure 103 shows plots of the lung burdens of diesel soot obtained in these experiments. Clearance from the low-level exposure group had a half-time of 87 ± 28 days, from the medium level exposure group of 99 ± 4 days and the high-level animals 165 ± 8 days. The half-time elimination figure for the high-level exposure group was significantly longer than for both the medium- and low-level groups. There was no significant difference in the half-times between the low- and medium-exposure groups.

It is clear from the results of this study that high-level exposures to diesel particulate (in this instance 4100 mcg/m^3) produces proportionately higher lung burdens of particles than would be predicted. This finding is in keeping with reports from the GM research group as well.

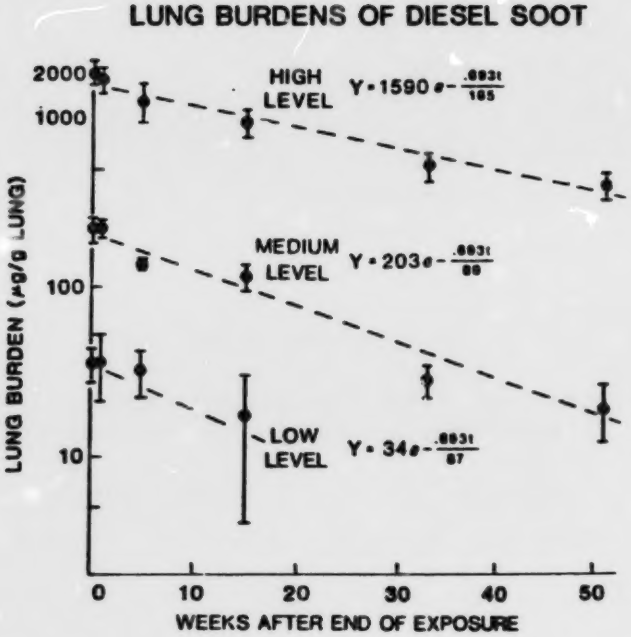


FIGURE 103

(Source: Reference 468)

In rats the total deposition was estimated to be about 25%, with lung deposition of around 15%. Table 108 shows the tissue distribution of radioactivity of $^{67}Ga_2O_3$ particles immediately after a one-day exposure to 0.1 um particles. From the data it was possible to estimate the retention times after varying periods of chronic diesel exhaust exposure (Table 109). After 10 days, the relatively insoluble particles will be predominantly in the lung. The whole body retention of the aerosols can be seen in Figure 104.

TABLE 108

Regional Deposition in Rats One-Half Hours After a 1-Day Exposure to 0.1 um Ga_2O_3 Particles

Tissue	Percent Sacrifice Body Burden
Lung	22
Gastrointestinal	27
Pelt	46
Head and Nares	1

(Source: Reference 258)

TABLE 109

Estimates of Quantities of Particles (mcg) Deposited and Retained in Rats for Three Exposure Levels

	4500 mcg/m^3			1000 mcg/m^3			200 mcg/m^3		
	Deposited		Retained	Deposited		Retained	Deposited		Retained
Exposure Time	WB[a]	Lung	Lung	WB[a]	Lung	Lung	WB[a]	Lung	Lung
1 Day	252	76	76	56	17	17	18	5	5
1 Week	1,260	378	272	280	84	60	88	26	19
1 Month	5,040	1,510	881	1,120	336	196	352	106	62
1 Year	65,500	19,700	3,640	14,600	4,370	809	4,570	1,370	254

[a] WB = Whole-body

(Source: Reference 258)

Wolff and Gray (260) reported mucociliary clearance measurements in rats after 1, 6 and 12 weeks of exposure during which a ^{99}Technetium-macroaggregated albumin suspension was instilled. The exposure used was the same as in the study described above (258). The results indicated a dose-related impairment in tracheal mucociliary clearance. There was a speeding up in clearance after one and six weeks in the high-dose animals which was thought to reflect a response of the system to clear the large number of particles

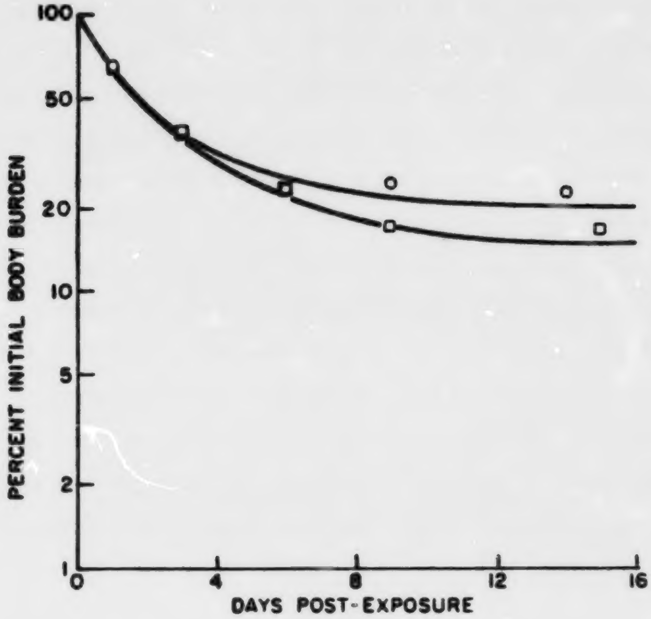


FIGURE 104

Whole-Body Retention of 0.1 um $^{67}Ga_2O_3$ Aggregate Aerosols in Rats after 1-day Exposure (□) and After 3 Days Exposure (○)

(Source: Reference 258)

deposited in the lung. Scanning electron micrographs showed minimal changes in ciliary morphology, but there was a tendency towards a lower percentage of ciliated cells in the medium and high-level animals.

Bacterial colonization and clearance patterns were studied in animals exposed for 12 weeks to the same amounts of diesel exhaust. Animals were inoculated with Pseudomonas aeruginosa and the pharyngeal area swabbed at various times after inoculation to determine colonization. Mice were exposed to aerosols containing ^{33}P-labelled P. aeruginosa. Half the animals were killed at 0 and half at 4 hours after exposure and the lungs removed, homogenized and quantitated using bacterial colony counts. An aliquot of the homogenized tissue was counted for radioactivity. It was found that diesel exhaust had no effect on the bacterial colonization rates of rats or mice. There was also no demonstrable effect of the exposure on *in situ* killing of bacteria. There was no significant difference in the amount of labelled bacteria present in the lungs of high-level exposed mice at 0 and 4 hours after inhalation of the labelled bacteria. There were significant reductions in the amount of label present in the lungs of low and medium-level exposed and control mice. The findings suggest that bacterial clearance was reduced in the high level exposure group, but calculated clearance values did not support that conclusion. The results suggested that exposure to these doses of diesel exhaust did not impair the bactericidal mechanisms of rodent airways to the respiratory pathogen *P. aeruginosa*. However, exposure to high levels of diesel exhaust may impair mechanical clearance mechanisms.

In additional experiments, the effects of inhalation of diesel exhaust particles on the immune and filtering functions of the lung-associated lymph nodes was evaluated (260). Exposed and control rats were immunized at 2, 5 and 11 weeks after the start of exposure by intratracheal instillation of sheep red blood cells. The numbers of lymphoid cells producing IgM anti-SRBC antibody were determined in the lung-associated lymph nodes and spleen 7 days after immunization (3, 6 and 12 weeks). The results showed that inhaled diesel particulates were cleared through the lung-associated lymph nodes and it was concluded that minimal or no effects on the induction of immunity after lung immunization were produced by the exposure levels and times included in this study.

Baseline respiratory function measurements were also performed on 20 male rats during the week prior to initiation of exposures. Ten of the rats were exposed to the highest level of exhaust and 10 were chamber controls. Animals were retested after 6 and 12 weeks of exposure and were killed for morphological evaluation after the 12 week measurements. Breathing patterns and dynamic lung mechanics were measured during spontaneous breathing. Subdivisions of lung volume, pressure volume curves, forced expirograms and CO-diffusing capacity were measured during hyperventilation induced apnea by inducing lung volume changes, either manually or with a positive-negative pressure respirator. The diesel exhaust exposure used in the study did not significantly alter respiratory function in vivo. Excised lung compliance was lower in high-level exposed rats, but the significance of this finding was unknown. There was no significant difference in the excised lung volume or weights between treated and untreated animals.

Twenty-nine rats were evaluated for histological changes resulting from 12-week inhalation exposure to diesel exhaust emissions. Only subtle changes were observed in rats exposed 12 weeks to the low level of diesel exhaust emissions. Although there appeared to be no absolute increase in pulmonary macrophages, those present contained distinct small black particulate cytoplasmic inclusions. In most instances the macrophages were either in alveolar spaces or occasionally on the surface of airways. Diesel particles were rarely seen in either interstitial macrophages or in bronchial-associated lymphoid tissue. Animals exposed to the medium diesel exhaust dose showed an increase in pulmonary macrophages and distinctly more phagocytozed particles (231). Although most were located within alveolar spaces, particle-laden macrophages were occasionally observed in the interstitium adjacent to bronchioles and within bronchial-associated lymphoid tissue. Rats exposed to the high exhaust level had considerably more phagocytozed particles within alveoli and bronchial-associated lymphoid tissue and in interstitial tissue. There was a marked increase in the number of pulmonary macrophages and their cytoplasm was packed with aggregated black particles.

In summary, the most significant observations consisted of a dose-related increase in pulmonary macrophages and in diesel particles present within macrophages. The deposition and retention of such particles over a period of 12 weeks did not result in morphologic alterations in either alveoli or small airways. At the highest level only, there was a suggestion of decreased tracheal goblet cell storage of mucous. Remarkably, little particle deposition was noted in bronchial-associated lymphoid aggregates.

During investigations of inhaled diesel particles at the General Motors Biomedical Research Lab, it was noted that the majority of the inhaled particles deposited in the respiratory system were removed from the alveolar and bronchial regions by several mechanisms which have been labelled collectively as lung clearance (4, 261). As has been discussed earlier, the major mechanism of lung clearance is the action of the cilia in the bronchial tubes moving the bed of mucous towards the oral pharyngeal cavity. On this mucous blanket are the scavenging alveolar macrophages which have picked up particulate, bacteria, etc., which are moved up to the pharynx, after which they are either coughed out or swallowed into the stomach.

However, there is an equally effective system of lung clearance in the lymphatic system which has been only moderately well investigated.

Since diesel particulate is often in the sub-micron size, aerosols frequently penetrate deeply into the respiratory system of man and animals. The ease of study can be promoted because the insoluble carbon core of the diesel particles is readily identified in the pulmonary regions.

There is very little information available regarding the deposition efficiency of inhaled diesel particles. The model used in previous studies suggested that a total deposition of approximately 35% of diesel particles with a 0.2 micron mass median diameter could be expected to occur in the deep alveolar pulmonary region. The General Motors study (4, 261) used Fisher 344 rats and Hartely Albino guinea pigs for "nose-only" inhalation testing. Inhalation testing following nose-only inhalation of diluted diesel exhaust generated from diesel engines burning Type 2D diesel fuel containing either ^{131}barium-labelled barium dodecalbenzene sulfonate or ^{14}C-labelled N-hexadecane. Lung burden of radioactive barium was measured by external scintillation counting and the deposition of ^{14}C was measured in tissues and filter samples following oxidation of the material. Within 4 hours after exposure, the deposition efficiency was determined to be 15 ± 6% with the radioactive barium tracer compared to 17 ± 2% deposition of inhaled diesel particles in the lungs based on ^{14}C content 30 minutes following the end of exposure. It was found that about 40% of the initial deposition of barium was eliminated in the first 4 days in the feces via the GI tract and clearly demonstrated the removal of diesel particles by mucociliary clearance in the upper airways. Very little clearance was observed in the test animals between 4 and 20 days. Later, activity was identified in the skeleton, indicating that barium had leached from the particles in the lungs and it was therefore decided that barium was an unacceptable tracer for use in studies requiring long-term retention of diesel particles in vivo.

The retention of inhaled ^{14}C-tagged diesel particles in the lungs was measured up to 105 days after exposure. The percent retention of inhaled tagged diesel particulate is presented in Figure 105. The data indicate at least 2 distinct phases of exponential clearance of the particulate. Clearance half-times of one day and 62 days were found for mucociliary and alveolar clearance, respectively. A small fraction of the particles, about 6% of the initial deposition, was found in the mediastinal lymph nodes after 28 days, which demonstrated that the lymphatic system was also involved in the removal of diesel particulate from the pulmonary system.

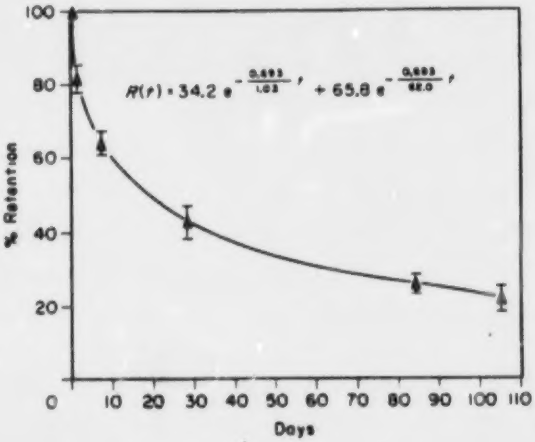


FIGURE 105

Retention of inhaled ^{14}C tagged diesel particles in Fischer 344 rats

(Source: Reference 4)

Animals were exposed to 250, 750 and 1500 mcg/m^3 of diesel exhaust for many different periods of time up to 45 weeks (261).

When the results were compared it was estimated that the maximum expected concentration in the lungs of experimental animals after one year for different levels of exposure indicated without doubt that, even for the lowest concentration of 250 mcg/m^3, the impact on the experimental animal was at least 1000 times greater than could be expected for a human population which would be exposed on the highways of our continent in the year 2000. The exposure estimate for man was based on the best estimate of the regional annual mean concentration of 3.5 mcg/m^3 for Chicago.

The administered dose apparently exceeded the capacity of an otherwise effective lung clearance mechanism which resulted in excessive accumulation of particles in the lung tissue and was easily recognized, even by visual examination.

However, under the microscope, obvious differences in the particulate deposits were seen in animals receiving high and low levels of exposure. It is important to note that in the early stages of low-level exposures, the scavenging function of the macrophages in the pulmonary tissue is so effective that free diesel particles in the lung parenchyma are rarely found.

The pulmonary alveolar macrophage is seen as the resident mononuclear phagocyte of the lung and is the primary defense against inhaled particulate matter in the lung. This important mechanism of cellular defense is not of local origin because recent studies with radioactive thymidine indicate that the alveolar macrophage is a monocyte that originates from the bone marrow and is transported in the form of a monocyte to the lung where it becomes a mixed or free cell able to phagocytose particles.

Because these alveolar macrophages are usually lying free in the cavity of the alveoli, they are easily washed out by bronchopulmonary lavage. The lavage fluid is centrifuged and the cells examined under the microscope.

Studies show that the inhalation of cigarette smoke induces increased DNA concentrations in lungs. This reaction is specific for pulmonary tissue and so it has been interpreted to indicate a mobilization and recruitment of the alveolar macrophages.

When the harvest of macrophages from the animals in the General Motors studies were examined, the motile size distribution in the control group showed a cell diameter of approximately 13 microns. At the exposure concentration of 1500 mcg/m^3 the macrophage population doubled and showed the enormous mobilization of the lung's defense mechanism. Analysis of the macrophages from the test animals demonstrated that there was another population of cells in the exposed group and these cells were only half the size of the normal alveolar macrophage, being about 8 microns in diameter.

With the help of special stains, the new cell population was shown to be distinctly different from the pulmonary alveolar macrophage. They showed cytology typical of neutrophils, which are a form of white blood cell. These neutrophils appeared to have phagocytic function as well because black particles could be seen in cells from animals in the advanced stages of diesel-exposure.

It should be noted that others have also observed the smaller cells when the animal has been stimulated by dust, other than diesel particulate. However, the smaller neutrophil cell is never present in very large numbers in a normal lung lavage fluid. In this test the specific role of neutrophils in initiating fibro-proliferative or inflammatory processes were not established and this aspect requires further study.

In human beings and animals, when phagocytosed particles are microbes, the macrophage has an effective system whereby enzymes are released to degrade the invading microorganisms.

Lymphatic fluid is produced on the terminal and peripheral aspects of the lung from the blood and is collected by lymphatic capillaries, and transported in a centripetal manner through peribronchial lymphatic channels towards the pulmonary hilus where it is filtered in the hilar and mediastinal lymph nodes before returning into the general circulation via the thoracic duct. However, this system rapidly changes when the cellular tissue defense is activated. In exposed animals in the laboratory the respiratory system shows alveolar macrophages, cells laden with particulates, found near the peribronchial and periarterial lymphoid aggregates. With continuing exposure the macrophages containing heavy loads of diesel particulate or free particulate aggregates were identified in large lymphatic vessels and lymphatic nodes. The lymph nodes can function as a very effective filter.

The solid black colour of lymph nodes from the pulmonary hylas and mediastinum indicates that there was an accumulation of diesel particulate during the exposure period.

It is reasonable to conclude that since similar findings have been seen in smoking populations humans exposed to other forms of dust, that these findings, which do not show further pathological changes in lymphatic tissue, indicate the extreme efficiency of the defense system of the organism. Obviously, the lymphatic system plays an important role in the clearance of inhaled diesel particles from the lungs and may be expected to provide significant protection in addition to the mucociliary clearance against the effects of inhaled diesel particulate.

The same GM group (262) reported on the clearance of ^{14}C-labelled diesel exhaust particulate by male rats after nose-only inhalation of a diluted exhaust from a single cylinder diesel engine. Animals were exposed to diluted diesel exhaust at particulate concentrations of 7,000 mcg/m^3 for 45 minutes and 2,000 mcg/m^3 for 140 minutes, resulting in similar total inhaled doses and comparable deposition efficiency. After exposure, the clearance of the radioactively-tagged particles was determined for extended periods of time. A plot of the particle-retention data (Figure 106) indicates a 3-component system with approximate half-lives of 1, 8, and 80 days respectively. The authors explained these half-lives in terms of clearance mechanisms. The first mechanism was considered to deal preferentially with particles deposited in the tracheobronchial tree and represented their rapid removal by the mucociliary escalator. These particles are eventually cleared through the GI tract and their elimination was clearly documented by the presence of ^{14}C activity in the animals' feces. The second mechanism was interpreted as the transport of material deposited in the most proximal respiratory bronchioles, where only a short distance is required for transferring the particulate to the mucociliary escalator. The third mechanism was thought to remove the particulate matter from the alveolar region, which may involve endocytosis, passive and active absorption and dissolution or metabolism. These studies are continuing and it is expected that they will provide further information on the presence of additional clearance phases of inhaled diesel particles and permit the assessment of their clinical significance.

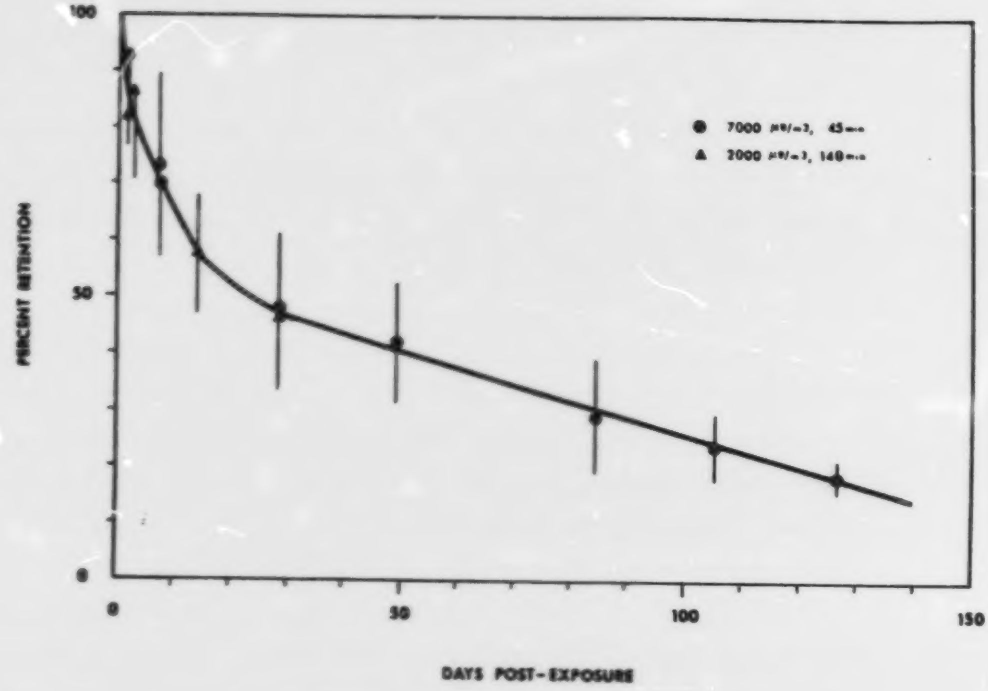


FIGURE 106

Clearance of inhaled diesel exhaust particles in Fischer 344 rats
The vertical lines represent standard deviations

(Source: Reference 262)

Strom and Garg (263) described the biochemical, morphological and physical measurements of the effect of diesel exhaust exposure at a particulate level of 250 mcg/m^3 for 6 months with serial sacrifice up to 16 months post-exposure in rats. The study was part of the previously described GM program in which animals were exposed for 20 hours per day, 5.5 days per week for 6 months. Light microscopic studies showed that immediately after exposure, diesel laden macrophages were diffusely distributed throughout the pulmonary alveoli, and also focally aggregated in some alveoli. Some macrophages containing diesel particulate could be seen in the bronchus-associated lymphoid tissue and in the lymphatics. Initially, 95% of the lavage macrophages were completely pigmented with phagocytized diesel particles. The percentages of these macrophages declined with time showing an exponential decay with a 6-week half-life. Macrophages were obtained in the same numbers as those from control animals and were of comparable cell size and had similar lysosomal enzyme activity.

With increasing post-exposure time, the diesel-loaded macrophages were no longer observed to be distributed diffusely throughout the lung, but remained, even after 16 months, in focal accumulations in the alveoli, particularly in the sub-pleural regions and in the lymphatics and bronchus-associated lymphoid tissue. The rapid decline of the diesel-laden macrophages, which was much faster than the overall diesel particulate removal from the lung suggested that, under conditions of prolonged exposure to high concentrations, disappearance of particulate-laden macrophages from the pool of cells which can be obtained by lavage seems to be due to the formation of aggregates of alveolar macrophages rather than transport out of the lung. The overall clearance of diesel particulate after extensive exposures appeared to be slow. After 16 months post-exposure, alveolar macrophages containing small amounts of diesel particulate were still identifiable in the lavage fluid. In addition, polymorphonuclear leukocytes were present among the aggregated macrophages suggesting that the incoming alveolar macrophages and polymorphonuclear leukocytes may have been involved in, or contributed to, the breakdown of the macrophage aggregates.

Chan and Lee (264) reported on differences in the deposition clearance of inhaled diesel exhaust particulates. Using ^{14}C-tagged particulate, it was found that the initial lung deposition efficiencies and mucociliary clearance half-times were comparable in both guinea pigs and rats. However, the amount of inhaled diesel particles cleared from the upper respiratory airways in the guinea pig accounted for only 17% of the initial lung burden, compared to 34% in the rat. Furthermore, the alveolar clearance of diesel particles in the guinea pig was extremely slow, with more than 80% of the initial dose retained after 105 days (Figure 107). The pulmonary clearance half-time for inhaled diesel particles in the guinea pig was estimated to exceed 300 days, which contrasted with the 60 - 80 days for the rat. This study clearly points out the difficulties in comparing studies of potential health effects of inhaled diesel particulate among different species and in extrapolating the experimental data to man.

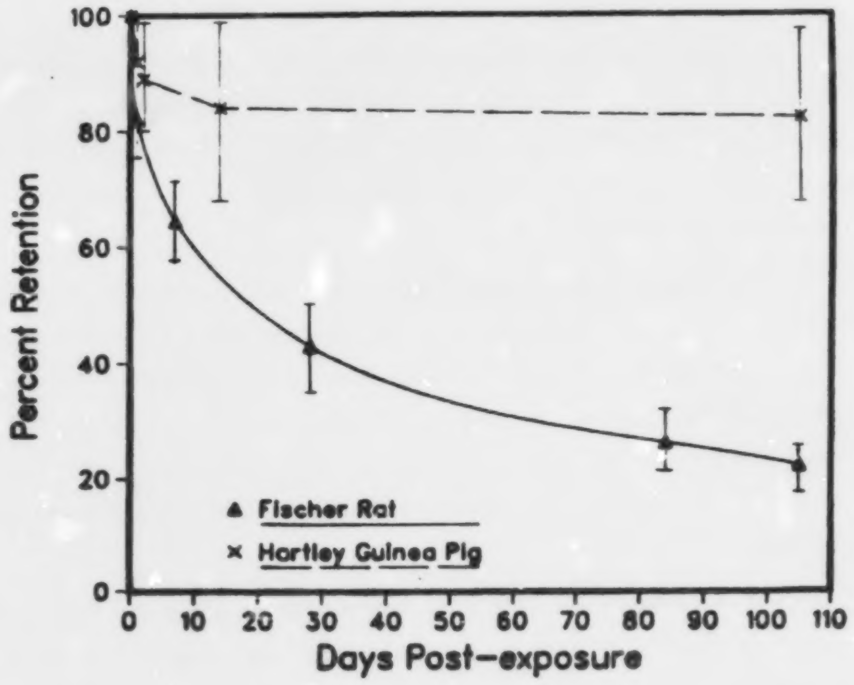


FIGURE 107

Lung Retention of Inhaled Diesel Particles

(Source: Reference 264)

As part of the GM program, Schreck et al (265) exposed juvenile male laboratory rats (Fisher 34 strain) and juvenile male guinea pigs (Hartley outbred albino strain) to diesel exhaust which was released as a fine aerosol with its primary route of entry into the body through the lungs.

Several levels of diluted diesel exhaust were used, including concentrations of 1500, 750 and 250 mcg/m^3. This proved the basis for a potential dose effect curve.

The lowest dose of 250 mcg/m^3, represented a more than 20 fold level compared to the ambient concentrations estimated to exist by 1985.

The emissions tested were from a 1978 light-duty passenger car (General Motors 350, 5.7 liter) diesel engine burning Amoco-2D Federal Compliance fuel.

The diesel engine was operated in a well-defined "cruise" manner since it is known that the physical and chemical properties of diesel particulate varies with engine speed and load.

Diesel aerosol mass concentrations had a variability of approximately 30% in each chamber where animals were kept, regardless of how the measurements and controls were regulated. This was seen largely as a mechanical limitation due to the equipment available at the time.

Throughout the experiment, attention was given to assuring that the aerosols delivered to the chambers were in the respirable range. If there was growth of particles in the atmosphere then they could not be expected to go deep in the lung. It was shown that the mass median aerodynamic diameter of the diesel particulate was 0.2 microns, with 90% of the mass associated with particles smaller than 1 micron.

The carbon monoxide (CO) concentration generally followed the trend of the diesel particulate concentrations over the time period monitored. The NO_X concentrations followed the pattern of the CO and the average ratio of NO to NO_2 was 94% to 6%. The NO_X

followed the chamber particle mass concentration in the same manner as carbon monoxide.

The GM group were cognizant of the fact that during whole body exposure of animals to diesel aerosols the animals intake of particulate occurs through feeding, grooming and breathing, in contrast to the miner where diesel exhaust intake is through the respiratory tract only. The dose received by way of all routes had to be known in order to understand the possible causes of any biological effects observed. The worst case estimate of the dose by feeding suggested it to be only 1/10th of the dose received through the respiratory tract.

Preliminary assessment of the data suggested that a greater amount of material was actually cleared through the GI tract as a result of grooming, than was deposited and retained within the pulmonary region. It should be remembered that the majority of particles and bacteria removed from the lungs is taken up by the macrophages and carried by the mucociliary escalator to the pharynx, at which point it is swallowed into the stomach.

Vostal et al (469) summarized the results of their diesel particulate deposition studies by calculating that about 8% deposited in the head of rats and rapidly entered the GI tract with another 6 -8% depositing in the tracheobronchial region of the lung and which was cleared through the GI tract with a half-time of about one day. A further 12% deposited in the alveolar regions of the lung. The diesel particulate in the latter area is quickly engulfed by pulmonary macrophages and in mice they estimated a half-time of phagocytosis of one day. Some of the scavenger cells containing particulate moved to the ciliated airways and are subsequently swallowed, while others are carried through the lymphatic system to the lymph nodes. They estimated the overall clearance half-time from the pulmonary region to be 60 - 80 days in rats. They also noted that after excessive exposures, some particle-laden macrophages did not clear the lungs, but aggregate in pulmonary connective tissue or in lymphoid foci in the lung parenchyma and in complexes near the terminal bronchioles.

In a second review of GM's work on diesel particulate and the pulmonary defense system (470), based on work on lung lavage experiments following exposure to diesel particulate, it was reported that at high exposure levels there are two immediate responses. One is a focal proliferation of alveolar Type II cells and the second is an increase in the number of phagocytosing macrophages. Interestingly, the high dose exposure in the GM studies was 1.5 mg/m^3, similar to that considered to be "typical" for the dieselized mine atmosphere. They also noted that only after "extreme levels of exposure" that focal accumulation of macrophages occurs which leads to an increased local deposition of cholesterol as intraalveolar deposits and which provoke local "quasi-pathological cellular changes resulting from the exaggerated protective response".

Rudd and Strom (492) developed a method to quantitate the particulate load in animal lungs using light attenuation of diesel particulate in aqueous solutions and suspensions of lung tissues. The animals used in validating the method were those exposed in the GM Inhalation Program and were exposed to average concentrations of diesel particulate of 269, 813 or 1530 mcg/m^3 for six months. There was a concentration-dependent increase in the amount of diesel particulate accumulated in the lungs (Figure 108).

Brooks et al (267) attempted to answer two important questions related to the potential carcinogenic propensity of diesel particulate, including; whether the particulate matter to which the carcinogenic material adsorbs is deposited and retained in the respiratory tract; and if the particulate is deposited in the lungs, are the carcinogenic compounds bioavailable to the pulmonary tissue so that it can exhibit its carcinogenicity?

The first point was examined in beagle dogs by measuring the pulmonary deposition of an aggregate aerosol of $^{67}Ga_2O_3$ with size and shape characteristics similar to diesel exhaust particles. As well, diesel particulate was incubated in various biological fluids and the extracted mutagenic activity determined and compared with the mutagenic activity of diesel particulate extracted with dichloromethane, which provided two fractions, one with a high and the other with a low mutagenic activity.

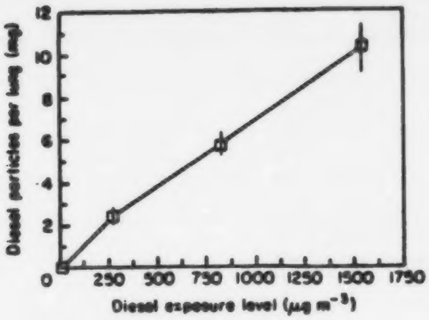


FIGURE 108

Accumulation of diesel particulates in guinea pigs exposed to diesel exhaust. Guinea pigs were exposed 22 weeks to 0, 269, 813 to 1530 ug/m^{-3} of DP. The amount accumulated in their lungs was measured as described in the Experimental section. Each point represents the average of duplicate analyses of three animals, and the standard deviation is indicated by the vertical line.

(Source: Reference 492)

Electron photomicrographs and measurements of aerosol properties, including count, median diameter etc., showed that the diesel aerosol and the surrogate aerosols were similar in size, shape and aerodynamic properties and both were small chain and cluster aggregates. The inhalation studies indicated that 33% of the inhaled material was deposited in the respiratory tract of the dog. Of the particles deposited, 82% were in the lung and of that in the lung, 63% was in the deep alveolar portions. Twenty-three percent (23%) of these particles were cleared rapidly with an effective half-life of less than one day. Additional studies are required to determine the long-term retention pattern.

Dichloromethane was the solvent most effective in extracting mutagenic activity from diesel particulate. However, a very small amount of mutagenic activity was removed by serum and lung lavage fluid over a 3-day incubation period, but no activity was detected when extraction was carried out with other solvents such as albumin, saline or dipalmitoyl lecithin. It was not clear as to whether the lack of activity was due to the fact that the mutagenic materials were not removed from the particles or whether they were bound within the extracting fluid and thus not biologically available.

Lakowicz et al (268), using fluorescent microscopy, measured the exchange of polynuclear aromatic hydrocarbons between particulates, such as silica or porous glass beads and lipid vesicles. They found that chrysene entered phospholipid bilayers much more rapidly from silica than as a microcrystalline particle. Transfer of chrysene from silica to the lipid vesicles was complete in 30 minutes. This research provided suggestive data that particulate and PNA's may be synergistic.

Following their original work, Lakowicz and co-workers (269) further investigated the uptake of benzanthracene from crystalline, microcrystalline and silica-absorbed states into lipid vesicles as a method of investigating the potential co-carcinogenic effect of particulate matter. As shown in Figure 109, the silica adsorbed state was more readily transported to dipalmitoyl-phosphatidyl choline vesicles than from the other states investigated. Carcinogen in the adsorbed state was more available than from crystals that were prepared by either solvent evaporation or sonication. These data were considered to be of possible significance in explaining the enhanced carcinogenicity of PAH's in the presence of particulate matter. It was suggested that the data might stimulate interest in the importance of PAH's in mines with rock formations high in silica content.

In a review of the effect of airborne and injected particulates as human carcinogens, Lakowicz (270) noted that most types of particulate contamination can greatly enhance the carcinogenicity of BaP, other polynuclear aromatic hydrocarbons, and systemic carcinogens, such as N-nitroso compounds. He also pointed out that the weight of evidence suggested that exposure to particulate contamination, even after carcinogen exposure had ceased, can pose a serious health risk resulting from irritation co-carcinogenesis. He

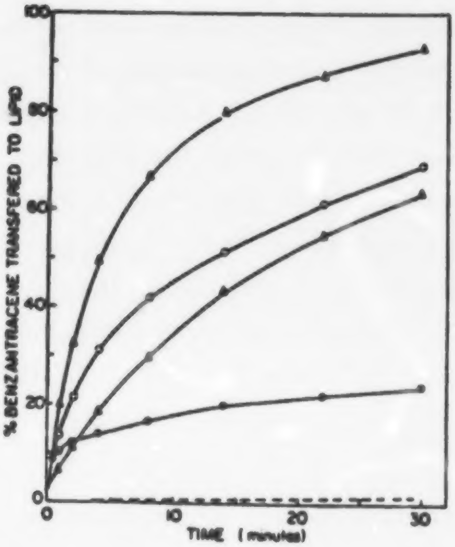


FIGURE 109

Membrane uptake of 1,2-benzanthracene. The percentage of benzanthracene transferred is shown for unsonicated crystals (------), sonicated crystals (●——●), sonicated crystals after benzene evaporated (▲——▲), and silica-adsorbed benzanthracene (△——△). The silica-adsorbed data is the average of the of the last five samples listed in Table 1 (in original paper). Also shown is the uptake data for sonicated crystals formed from benzene evaporation to which 10 mg of silica was added after sonication (○——○). The amounts of benzanthracene varied from 6 to 30 ug, and the amounts of dipalmitoyl phosphatidylcholine vesicles from 4 to 15 mg. The dipalmitoyl phosphatidylcholine to benzanthracene weight ratio was 500.T = 31°C.

(Source: Reference 269)

suggested that particulate contamination can result in the expression of pre-cancerous lesions which are already present in the body, or can enhance the carcinogenicity of agents to which we are commonly exposed.

In an extension of their earlier work, Lakowicz and co-workers (271) investigated the absorption of BaP to particulate matter and its rate of uptake into model membranes. Again they demonstrated that adsorption to a variety of non-fibrous particles including silica, resulted in increased rates of membrane uptake when compared with aqueous suspensions of BaP microcrystals. BaP, however, was not released from carbon black during these experiments. Adsorption of BaP to the surface of the particles was necessary for the enhanced transport of the PAH into membranes. The rate-limiting step for the transport of BaP into the lipid vesicles appeared to be its rate of desorption from the surface of the particle, after which subsequent membrane uptake of BaP was rapid. These in vitro experimental models were considered to help in the understanding of the fate of particle-adsorbed BaP in the lungs, thus providing clues as to the environmental cause of lung cancer in humans. Fibrous materials, such as asbestos, had the best ability to absorb BaP in the monomeric state and was most rapidly desorbed and taken up by the phosphatidylcholine vesicles.

Lakowicz and Bevan (272) noted that PAH by itself, after inhalation or instillation in lung tissue, causes a low incidence of lung cancer unless particulates are also instilled. It is not known if particulate adsorbed PAH is eluted directly into lung surfactant, with subsequent transfer to cells, or if the PAH is eluted after phagocytosis. However, regardless of the site of elution, particles which rapidly release adsorbed PAH could increase the effective dose of carcinogen in the lungs by elution of these compounds prior to clearance of the particles from the lungs. Phagocytosis of particles with adsorbed PAH could increase the intercellular availability of the PAH from microsomal activation, which is often required prior to their becoming carcinogenic.

As pointed out by Tyrer et al (273), the carcinogenic potential of diesel engine exhaust can be in part evaluated by examining the fate and distribution of PAH's in animals exposed to emissions. It is believed that the mechanism for carcinogenesis due to PAH's follows the steps shown in Figure 110. The PAH is absorbed and metabolized by some, if not all, tissues. As a result of enzymatic oxidation reactive intermediates are produced which are conjugated to form water-soluble metabolites which are then excreted. The reactive intermediates may bind to cellular macromolecules leading to cell death or tumour formation. This group studied the metabolism of BaP after intratracheal instillation of a radio-labelled compound to mice.

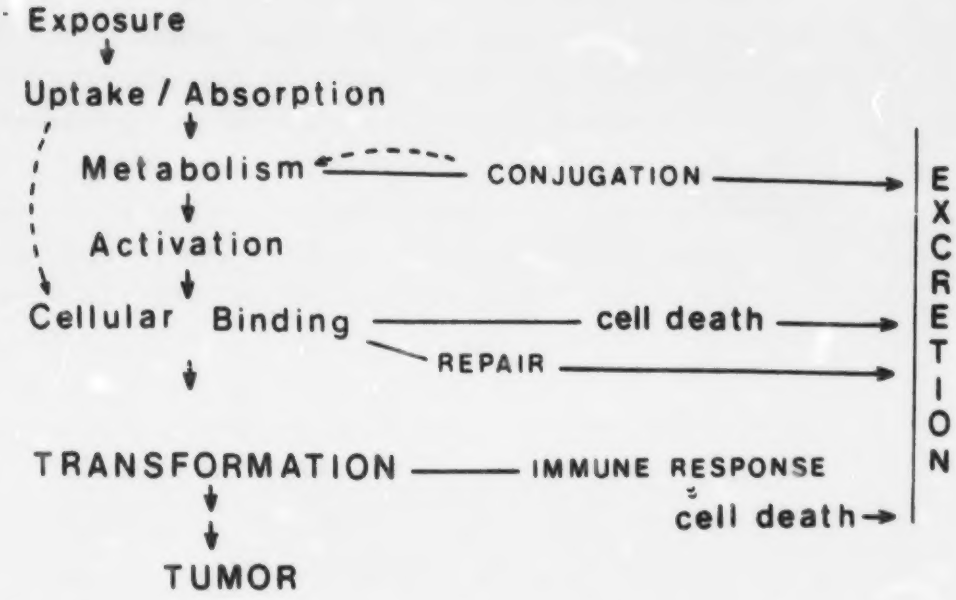

Exposure
Uptake / Absorption
Metabolism
CONJUGATION
Activation
Cellular Binding
cell death
REPAIR
TRANSFORMATION
IMMUNE RESPONSE
cell death
TUMOR
EXCRETION

FIGURE 110

Carcinogenesis Pathway for Polynuclear Aromatic Hydrocarbons

(Source: Reference 273)

Histofluorescent examination of tissues after instillation of non-radioactive BaP confirmed that the compound was present in lung tissue. With radioactive BaP radioactivity was detected in the entire animal within 2 hours, with most of the activity being in lungs, liver and GI tract. By 24 hours considerable radioactivity had redistributed to the GI tract, but by 168 hours after instillation, only a trace of label was found in the gut. The fluorescent data confirmed that the BaP was deposited in the bronchi of the lungs.

This same group (274) further examined the metabolism of BaP by measuring the content of free non-conjugated primary metabolites, sulphate conjugates, glucuronides, glucothiones and other conjugates in tissue extracts from similarly treated animals.

Two hours after instillation primary metabolites were present in liver and lung, but little of the material was conjugated. The unconjugated BaP was mainly in the form of free BaP and phenolic metabolites. During the first 16 hours after instillation, 14% of the BaP was excreted in urine by diesel exhaust-exposed mice, compared to [illegible] excreted by non-exposed mice. The only meaningful difference between the exposed and unexposed mice was a suppressed ability to clear the small amount of BaP from the lungs, which would suggest that BaP was adsorbed to smoke particles. In the lung, the majority of radioactivity was present as non-metabolized BaP. In contrast, liver and testes contained more than half of the radioactivity as metabolites, with a substantial amount of secondary metabolism to conjugates. As noted above, both groups were capable of clearing the bound BaP, but there was a significantly higher amount of residual BaP in lungs of exhaust-exposed mice one week after instillation.

Medda, Dutta and Dutta (275) measured the bioavailability of tritiated BaP adsorbed to diesel exhaust particulate after intratracheal instillation in female guinea pigs. The percentage of radioactivity retained by the lungs 20 - 25 minutes after instillation was 77 to 95% of the dose. Forty-eight hours following instillation there was an average disappearance of 42 ± 6% of the radioactivity. By 48 hours, the radioactivity had distributed widely into organs such as liver, kidney and intestines. Twenty-five percent of the radioactivity was excreted in the urine and feces of the animals by 48 hours. The studies showed that the BaP dissociated from the diesel particles after instillation in the lungs. However, the authors cautioned that the rapid dissociation from the diesel particulate implied that their method of labelling may not have simulated the in vivo situation of BaP binding to diesel particulates under engine conditions.

Farrer, Dutta and Dutta (276), determined the effect of diesel smoke on pulmonary protein synthesis in male rats exposed to 6 mg/m^3 of diesel particulate for 2, 4 and 8 weeks. Studies were carried out by way of lung perfusion in anesthetized animals, with the incorporation of tritiated leucine being measured by autoradiography and isolation of lung protein. At 8 weeks of exposure, there were no significant differences in the incorporation of tritiated leucine into the trichloroacetic acid insoluble protein. Similar results were obtained after 2 and 4 weeks of exposure to diesel exhaust. Electron microscopic autoradiographic grain counts from the diesel-exposed versus control rats showed no particular differences among the various groups.

Schneider and Felt (277) investigated the effects of 250 and 1500 mcg/m^3 of diesel particulate from diesel exhaust on the adenylate or guanylate cyclase activity obtained from liver and lung tissue of rats and guinea pigs. Soluble stimulated guinea pig lung guanylate cyclase was first increased at 6 weeks, decreased by 24 weeks and at 52 weeks there was no change. The basal activity of rat liver adenylate cyclase was generally unchanged throughout 52 weeks of diesel exposure. The findings suggested that diesel exposure did not substantially alter either of these intercellular regulating enzymes.

Eskelson and co-workers (278) administered diesel particulate intratracheally to rats and then measured the lipid content of lung lavage fluid, including the incorporation of ^{14}C-acetate into fluid lipids. Five days after administering the diesel particulate, a threefold increase in palmitic and arachidonic acids were measured. Stearic, oleic and linoleic acids were not significantly changed. The study suggested that the lipid loading observed in lungs exposed to 5 mg of diesel particulate for five days, was a result of the increased deposition of pulmonary surfactant or extracellular lipids, and not as a result of intracellular lipids. As well, the phospholipid and cholesterol content of the lungs was significantly increased, whereas triacylglycerols were not changed (266). At the same time, hepatic phospholipids and cholesterol levels decreased. Further studies suggested that a pulmonary lipotropic factor exists which stimulated lipogenesis in the liver, leading to denovosynthesis of lipids which are used to maintain lipid homeostasis in the lung.

Navarro, Charboneau and McCauley (279), as part of the GM Diesel Exhaust Inhalation Program, measured the ability of the polycyclic hydrocarbons in diesel exhaust when administered by inhalation to either locally induce the microsomal oxidases in the lung and/or affect liver enzymes as well. They failed to measure any effect of diesel exhaust exposure for a year on the ability of liver microsomes to oxidize benzo(alpha)pyrene to more polar metabolites. Furthermore, it was found that the liver microsomes, after several months of exposure, did not induce either cytochrome P-450, cytochrome P-458 or NADPH-dependent cytochrome C reductase. The data for rat lung microsomal oxidation of benzo(alpha)pyrene was suggestive of an inhibition by 24 weeks of exposure to 1500 mcg/m^3 of exhaust. Similarly after 53 weeks of exposure to 250 mcg/m^3 of diesel particulate, lung microsomal activity was reduced compared to control animals. Exposure to an intermediate concentration of 750 mcg/m^3 for 24 weeks also seemed to impair the ability of lung microsomes to oxidize the BaP.

Based on previous reports that guinea pig pulmonary prostaglandin dehydrogenase (PGDH) is altered by acute exposure to NO_2 for short periods of time, Chaudhari, Farrer and Dutta (493) investigated the effect of long-term exposure of guinea pigs and rats to diesel exhaust on PGDH pulmonary activity. Given that PGDH is an important regulatory enzyme present in high concentrations in lung tissue, it was thought that this enzyme might serve as a biochemical marker for adverse effects of environmental toxins upon the lung. At the lowest dose of diesel exhaust (250 mcg/m^3 of particulate), there was a large, significant increase in PGDH activity compared to matched controls after 1.5 months of exposure. This increase was not seen in animals exposed to 1500 mcg/m^3 of diesel particulate. At the high dose there was, however, after 3 and 6 months of exposure a time-dependent decrease in enzyme activity. It was noted that the rat was not a good species for examining this effect as the enzyme level was only 10% of that in the guinea pig. There was a large age-dependent increase in enzyme activity between 6 and 12 months in both the control and exhaust-exposed animals.

Cyclic nucleotides are products of tissue adenylate cyclase or guanylate cyclase systems and their activity is altered by various hormones, neuro-transmitter substances, drugs, etc. As well, it has been shown that the enzyme activities may be altered by inhalation of toxic products or in experimentally-induced neoplasias. Schneider and Felt (280) examined the effect of 250 or 1500 mcg/m^3 of diesel particulate administered to rats and guinea pigs by inhalation on lung and liver adenylate and guanylate cyclase activity. Recently, the urinary excretion of cyclic nucleotides has been found to be increased in animals with benign or neoplastic tumours and other in vitro studies have shown both cigarette smoke and nitrous oxide to maximally activate guanylate cyclase from rat liver. The investigators failed to find any consistent change in adenylate or guanylate cyclase activity compared to control animals for either lung or liver enzyme activities. They did, however, note an age-related decrease in total adenylate cyclase activity in rat liver which may have been stimulated by diesel exposure at 3 and 12 months. As well, a biphasic exposure related response of guinea pig liver, soluble guanylate cyclase through week 24 of the study was noted and an increase in adenylate cyclase basal activity in the rat lung occurred for the 52-week duration of the study but was unrelated to diesel exposure.

4.5.2 Mutagenicity of Diesel Exhaust Using In Vitro Bacterial Test Systems

Although the following sections are organized so as to differentiate between bacterial (Section 4.5.2) and mammalian (4.5.3) test systems, overlap does occur, particularly where investigators have employed a mutiplicity of methods. For reasons of coherence, all of the results of a particular study are discussed together, which may include results from both bacterial and mammalian test systems.

The mutagenicity testing of compounds using in vitro microbiological assays depends on the use of up to five mutant strains of Salmonella typhimurium. Each strain of bacteria contains a mutation in its gene coding for histidine biosynthesis, which makes the organism unable to grow on a media which does not contain histidine. In addition to the histidine mutation, each strain has a structural modification such that agents may permeate more easily into the bacteria or interfere with the bacteria's genetic repair mechanisms. These features permit the action of a mutagen to be expressed and thus detected by the assay. Exposure of the mutant strains to compounds which can alter their genetic activity results in some of the mutant cells reverting to their wild-type state, which involves regaining the ability to synthesize histidine. These organisms are demonstrable as colonies on histidine-free media (281, 282, 471, 472, 473).

Purchase et al (474, 475) evaluated the reliability of 6 short-term tests in predicting carcinogenesis of 120 compounds. They reported a 91% correlation between the Ames test and carcinogenicity with 58 carcinogens and a 93% correct prediction between mutagenicity and carcinogenicity with 62 non-carcinogenic compounds. The cell transformation assay, using the Syrian hamster kidney fibroblast, had a 91 and 97% accuracy, respectively. Other tests, such as the loss of ribosomes from rat liver, endoplasmic reticulum following incubation with carcinogens and the implant test and sebaceous gland test, etc., were less accurate.

In some instances, investigators have used all five Salmonella typhimurium strains in their testing, but in many instances, only one or two strains, particularly TA-98 and TA-100 are used, because these strains appear to be the most responsive to the organic compounds extracted from diesel exhaust particulate.

However, as was pointed out by Claxton, mutagens that cause base-pair substitution may be overlooked if strain TA-1535 is not employed. By not using strain TA-1538, indirect-acting mutagens which require activation by addition of an exogenous, microsomal enzyme system could also be missed.

Enzyme systems obtained from mammalian microsomal liver fractions (called S9) are often added as a part of the test system. Many substances are not mutagenic outside the animal system but are structurally altered to a mutagenic form by microsomal enzymes within animal tissues. Thus an exogenous microsomal system may be added to ensure the full expression of the mutagenic potential of a compound. The S9-system may also structurally change a direct-acting mutagen into a non-mutagenic form, thus putting its mutagenic significance in doubt because of its possible in vivo inactivation. The number of colonies induced by mutagenic substances at a given dose provides a measure of the potential hazard of the substance (281, 282).

In reviewing the results of mutagenic testing, an activity which has proliferated rapidly over the past few years, it should be kept in mind that many experimentally induced effects can occur, such as temperature changes and oxidation, which can alter the mutagenic activity of an extract. Further, the type of solvent used to extract the particulate matter and the solvent-soluble components also results in differences in activity in mutagenic tests. As detailed later, Siak (283) showed that when diesel particulate was extracted with 0.5% bovine serum albumin, simulated lung surfactant or saline, no mutagenic activity could be detected. Only fetal calf serum and organic solvent extracts, such as DCM (dichloromethane), had mutagenic activity. The results indicate that the mutagens attached to diesel particulate might not be readily available in vivo, thus putting in doubt the mutagenic significance of diesel particulate extracts.

Epler (476), in a comprehensive review of the use of short-term mutagenic tests, cautioned against using them as predictors of carcinogenesis. He emphasized that extrapolation to a relative biohazard could be premature. The detection or perhaps the generation of mutagenic activity may be a function of the chemical fractionation system used. Both false positives and false negatives could occur and the choice of inducer could be wrong, particularly if the liver contained both activating and deactivating enzymes. He pointed out that the correlation between mutagenicity and carcinogenicity still required correlating in suitable test models.

The cautions are not, however, to be taken to mean that the short-term microbiological mutagenicity assays are not of substantial value.

Clark and colleagues (313) examined the mutagenic activity of diesel exhaust particulate extracts from a variety of small diesel engines used for passenger vehicles and concluded that the similar mutagenic potency of extracts from the 6 different diesel engines operated on the same fuel suggested that engine design had very little influence on the mutagenicity of the particle-associated organic compounds. There was no significant difference in the mutagenicity based on driving cycle variations as well. However, there was a large variability in the extractable fraction and particulate emission rates between the different engines and even in the same engine operated on different cycles which emphasized the need to include these variables when estimating the quantities of potentially hazardous materials emitted in the exhaust.

Siak emphasized that the bioavailability of mutagens attached to diesel particulate must be considered in the final assessment of any potential effect of diesel particulate in biological systems and organisms.

The Ames test is a test of mutagenicity to bacterial cells. Of more importance may be the mutagenicity of extracts employing mammalian and human cell lines.

In this vein, Zweidinger (4, 284) reviewed the many variables which can affect mutagenicity testing such as sampling procedures, sample preparation, engine differences and operational characteristics.

Clark and colleagues (285) examined the mutagenic activity of diesel exhaust particulate extracts from a variety of small diesel engines used for passenger vehicles. They concluded that the similar mutagenic potency of extracts from 6 different diesel engines operated on the same fuel suggested that engine design had very little influence on the mutagenicity of the particle-associated organic compounds. There was no significant difference in the mutagenicity based on driving cycle variations. There was a large variability in the extractable fraction and particle emission rates between the different engines, and even in the same engine operated on different cycles, which emphasized the need to include these variables when estimating the quantities of potentially hazardous materials emitted in exhaust.

Huisingh and co-workers (286) collected particulate samples using dilution tunnel sampling techniques. The emission samples were Soxhlet-extracted with DCM to remove organics. DCM was found to be the most effective of a series of solvents in removing genetically-active organics from diesel particles. The Soxhlet-extracted organics were filtered using Teflon millipore filters to remove particles. Large volumes of solvent were concentrated by rotary evaporation under reduced pressure. Aliquots were evaporated to dryness under nitrogen and stored frozen in the dark, until tested for mutagenicity.

Engines used included Caterpillar, Nissen, Oldsmobile and VW Rabbit diesels as well as a gasoline engine from a Ford Mustang. Except for the Caterpillar engine, all were light-duty motors. Cigarette smoke condensate and known mutagens, such as BaP, were included in the study.

Mutagenicity tests in bacterial, yeast and mammalian cell systems were selected to demonstrate gene mutations, chromosomal effects and DNA damage. The results of these mutagenic studies were reported in a series of articles (287, 288, 289, 290, 291).

Samples from the various combustion sources were demonstrated to be mutagenic in the Salmonella typhimurium assay (287). Cigarette smoke condensate required activation before mutagenic activity was seen, whereas most of the activity associated with diesel samples was direct-acting and did not require activation. There was a lack of response in Salmonella strain TA1535, which suggests that most, if not all, of the activity was due to polynuclear mutagens and not to alkylating agents. A maximum difference of about 25-fold was measured between the Caterpillar and Nissen diesel samples.

Castro et al (289) examined extracts of diesel and gasoline engine emissions and compared them to extracts of roofing tar and cigarette smoke condensate in a viral enhancement assay.

All samples produced a positive dose-response. The emission extract prepared from the Caterpillar heavy-duty engine failed to induce a significant increase in the adenoviral transformation frequency. Only four samples caused a significant dose response increase in 6-thioguanine resistant cells (Nissen, VW Rabbit, Mustang and coke oven). Induction of cellular DNA breakage appeared to be the least sensitive of the three assays. Only the Mustang and coke oven emission extracts produced a significant change in the sedimentation profile of the DNA from treated Syrian hamster embryo cells.

Using the Ames Salmonella microsome system, Ohnishi et al (292) found mutagenic activity in the emission exhaust from diesel engines, which were not typical of those used in mines. The samples were tail-pipe collections, which would not contain components typical of a ventilated mine. However, the study described the presence of various mutagenic polycyclic hydrocarbons in exhaust.

Brooks et al (293) found that the fuel sulphur content had little influence on the mutagenic activity of methylene chloride extracts of exhaust particulate from an automobile diesel engine operated without an oxidative catalyst. However, when the car was operated with a catalyst and low-sulphur fuel, the exhaust extract showed increased mutagenic activity. Operating the car with a catalyst and a high sulphur fuel resulted in a marked decrease in both the mutagenic activity per mcg of extract and per mg of particles emitted. The addition of the exhaust catalyst and the use of the higher sulphur fuel resulted in increased particulate emissions, which could have resulted from the conversion of SO_2 to sulphates. This may explain the decrease in the percent of the particulate material that was extracted from the filters with methylene chloride.

As noted by Henderson and colleagues (294), diesel exhaust particle extracts have both direct-acting mutagenic and cytotoxic properties, but the chemical nature of the biologically-active components is not well established. One of the mechanisms which has been proposed for the formation of direct-acting mutagens is the reaction of PAH's with NO_2/O_3 or other oxidative pollutant gasses. These researchers found that the reaction of diesel fuel aromatics with NO_2 resulted in the formation of nitro-aromatics. The starting fractions were inactive as mutagens but after reaction with NO_2 the products contained frame-shift mutagens which did not require activation by microsomal enzymes. The cytotoxicity induced by diesel exhaust was reversed by serum or glutathione. The LC_{50} of the diesel fuel fractions to Chinese hamster ovary (CHO) cells was increased 5-fold after reaction with NO_2.

Bradow and co-workers (295) completed a number of experiments in which NO_2 was added to the dilution air of diesel exhaust emissions to determine whether the NO_2 converted polynuclear aromatic hydrocarbons to biologically-active species, as determined by mutagenicity testing. Ames test specific activity and nitropyrene levels increased above baseline when the NO_2 introduced into the exhaust stream exceeded 5 - 10 ppm. Re-exposure of filter particles to diluted gas-phase diesel exhaust caused little increase in nitroaromatics or Ames activity.

Loprieno et al (296) published preliminary results of in vitro mutagenicity experiments on particles and four particulate DCM extract fractions. The assays were done using five strains of Salmonella and 2 yeasts. The two strains of yeast allowed the evaluation of the potential activity of samples for production of gene mutation and gene conversion in the diploid cell. A heavy-duty (sample A) and a light-duty (sample B) engine were studied. The extracts from these engines were used for the induction of unscheduled DNA synthesis (UDS) using a human cell line and in vitro tests were completed on other samples of diesel engine exhaust.

The diesel extract from the heavy-duty engine was tested for its ability to induce UDS in human cells. This provides information as to the direct reactivity of a chemical towards DNA by evaluating the repair-synthesis of DNA which might follow the induction of some type of damage to the DNA structure. There was an indication of a stimulation of unscheduled DNA synthesis.

Similar to other studies, particular methylene chloride extract fractions labelled PRF (paraffinic), ARM (aromatic), TRN (transitional) and OXY (oxygenated) were tested. The PRF and ARM fractions were negative in all test systems. The TRN was mutagenic to four strains of Salmonella. This fraction contained direct mutagenic chemicals, and in the presence of the S9 activation enzymes, showed an increase in activity. TRN was not mutagenic to the yeast S. pombe and it did not induce mitotic chain conversions in S. cerevisiae.

OXY fractions were found to be positive in all the Salmonella and yeast test systems.

In vitro diesel exhaust particles were mutagenic both in the presence and absence of S9 liver fraction. The TRN and OXY fractions represent 3.16% and 10.81%, respectively, of the total amount of diesel particles. The particles and the TRN and OXY fractions were compared in the same strains with the revertants being highest with the OXY fraction (6.7), midrange with the TRN (2.1) fraction, and lowest with the particles (0.7).

Using the Ames test, mutagenicity was demonstrated and shown to increase by the addition of S9, but the addition did not influence the mutagenic activity of the same B extract from the light-duty engine. The test for the induction of unscheduled DNA synthesis with the human heteroploid cell line indicated inactivity at levels below 1 mcg/ml, whereas it was extremely toxic to cell DNA replication at concentrations above 1 mcg/ml.

At concentrations greater than 1 mcg/ml, diesel particulate extract inhibited normal DNA replication, thus showing a direct effect of this fraction on DNA as being a type similar to other well-known DNA inhibitors.

The particles and the four fractions obtained from a neutrally-aspirated swirl chamber demonstrated mutagenic activity on TA98. The OXY fraction had the most activity in the absence of S9. The OXY fraction also had genotoxic activity in the yeast *S. pombe* (forward gene mutation system) and in *S. cerevisiae* (mitotic chain conversion system). This supports the hypothesis that the OXY fraction contains specific mutagenic chemicals.

Diesel exhaust particles from two different light duty diesel engines were extracted by Brooks et al (267) with organic solvents, such as methylene chloride (MC) yielding mutagenic activity in the Ames system. They studied the deposition and early retention of ultrafine particles with size and shape similarities to diesel exhaust particulate, and the biological availability of mutagenic compounds that are associated with the diesel exhaust particles in dogs (see section 4.5.1 for details).

A surrogate aerosol was used that was similar in size, shape and aerodynamic property to diesel emission aerosol and a significant portion was deposited in the deep alveolar portions of the respiratory tract. Twenty-three percent of the particles were cleared rapidly with an effective half-life of less than one day.

In addition to MC, particles were incubated in biological solvents, such as serum and lung lavage fluid for different times and at various particulate concentrations.

Two Salmonella strains, TA98 and TA100, were used in the Ames tests. When S9 was added, it decreased the magnitude of the response.

The mutagenic activity of the extracts did not change markedly as a function of the incubation time. The only biological solvent to show a positive response was serum, and it was very slight.

When tests were conducted using strain TA98, there was a high mutagenic activity in one sample. There was an apparent dose-relatd increase in mutagenic activity in the serum extract, with an indication that the lung lavage fluid could also remove a small fraction of the mutagenic activity. There was a certain number of mutagenic revertants in the MC, serum, and lung lavage extracts. Saline extraction failed to show mutagenic activity above that observed in the control cultures. The small particle aerosols resulted in a higher lung deposition in the dogs than reported for larger aerosols (1.8 microns). The data suggests that the majority of the aerosols are deposited in the lung region, where clearance is slow. The toxicological implications are that the lung retention time of particles needs to be considered when determining the biological availability of associated organic compounds.

An attempt was made to address the question of the chemical dose received by the lung by extracting the diesel particulate with a range of solvents similar to those found in the biological situation. The reversion response of bacteria using MC extracts of the two diesel particulate samples differed by a factor of about 3 for TA100 and 10 for TA98, without the addition of activating systems (S9). As discussed above, only a small amount of mutagenic activity was obtained with the biological fluid extracts. This may have been because the mutagens were not desorbed from the particles or because the biological fluids may have bound or inactivated the mutagenic components associated with the particulate, even after they were removed from the particles. Alternately, the bacteria may not have been able to incorporate bound mutagens and produce a positive response. However, in other studies, mutagens extracted from fly ash particles with horse serum produced mutagenicity in the Ames system.

The slopes of the dose-response curves can be compared for serum, lung lavage fluid, and the organic solvent MC (Figures 111 - 113). These indicate that the biological solvents extracted from 0 to 3% of the mutagenic activity extracted with CH_2Cl_2, as measured by TA100 without S9. For TA98, the mutagenicity for serum and lung lavage fluid were similar, having 10% of the response of the organic solvent extract. Extrapolation to whole animals is questionable. The studies were conducted over a short period of time and may not adequately reflect the solubility effects occurring during the longer residence times involved in lung clearance of particulate in animals and human beings. Secondly, the lung lavage fluid or serum may bind or detoxify mutagenic compounds and make them unavailable for interaction with the bacteria, even though they may be removed from the particles in vivo. Finally, the lung environment consists of cellular elements as well as fluids. The cellular elements may extract, detoxify or activate the mutagenic compounds in vivo, and thus the in vitro solvent systems may be less applicable to the whole animal situation and hence interfere with our understanding of the changes which occur in the lung.

Siak et al (283) also found that the mutagenic activity of diesel particulate was not readily extracted by simulated biological fluids. Nineteen and 90% of the mutagenic activity of the diesel particulate extract was in only two fractions consisting of polynuclear aromatic compounds.

No mutagenic activity was detected using 0.5% bovine serum albumin, simulated lung surfactant, or saline extracts, even at particulate concentration of 5 - 10 mg/ml. Only fetal calf serum extracted mutagenic activity from diesel particulate.

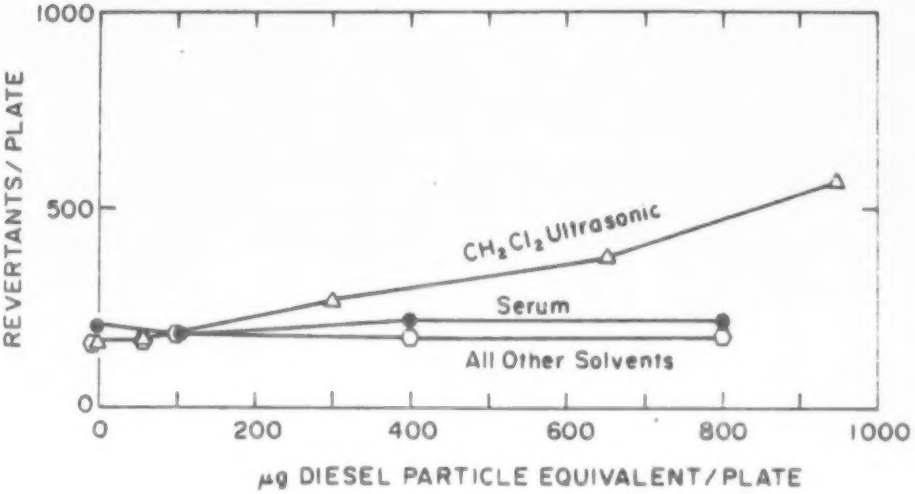


FIGURE 111

Dose-Response Relationship for Mutations Induced in TA-100 by Extracts From a Diesel Particle Sample 2 With a Low Mutagenic Activity

(Source: Reference 297)

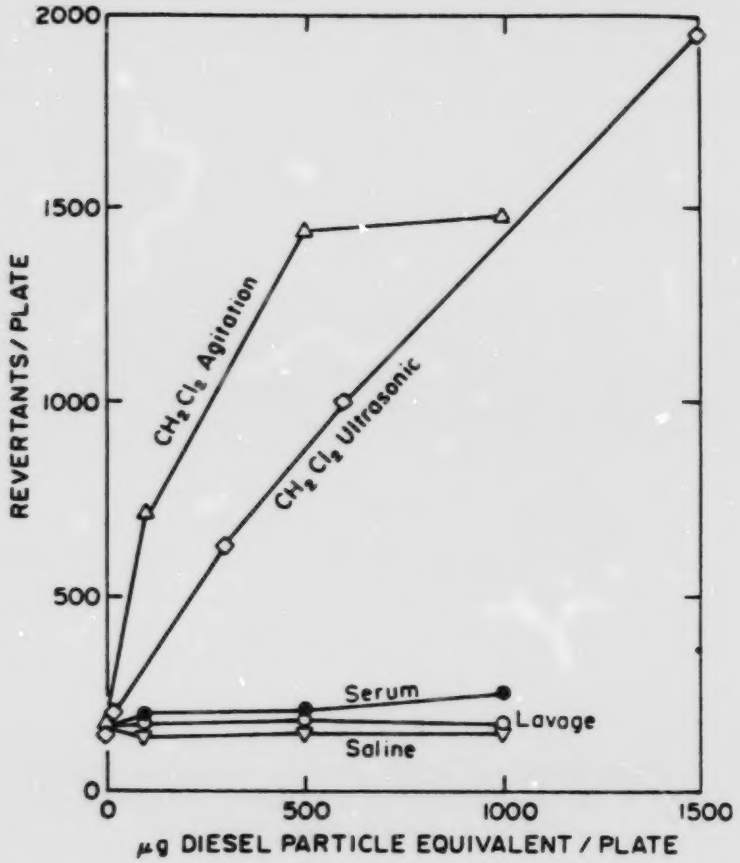


FIGURE 112

Dose-Response Relationship for Mutations Induced in TA-100 by Extracts of Diesel Particle Sample 1 With a High Specific Mutagenic Activity

(Source: Reference 297)

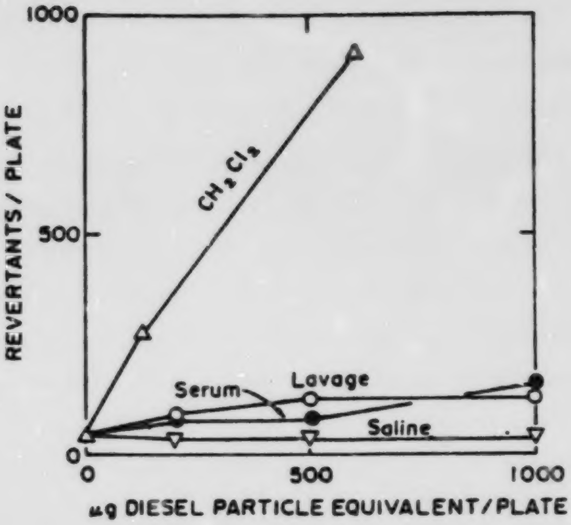


FIGURE 113

Dose-Response Relationship for Mutations Induced in TA-98 by Extracts of Diesel Particle Sample 1 with a High Specific Mutagenic Activity

(Source: Reference 297)

Choudry and Doudney (298) extracted particulates from light duty Volkswagen Rabbits and Mercedes 300D diesel engines. Extraction was with dichloromethane for 24 hours in a Soxhlet extractor, followed by fractionation by liquid/liquid partitioning to separate acidic, basic and neutral compounds using a solvent gradient of hexane, dichloromethane and diethylether for elution.

The VW Rabbit sample showed the neutral fraction comprised approximately 94% of the extractable mass.

The VW engine whole extract had mutagenic activity with all five strains of Salmonella. Activity was present in the absence of S9, indicating the presence of direct-acting mutagens. A positive response in all strains suggested that the mutagens affected both base pair substitution and frame-shift mutation.

The unfractionated extract from the Mercedes 300-D showed a high response in the Ames test with the strains TA98 and TA100. Again, activity without S9 was higher than with S9.

The specific activities of the 3 fractions compared to the crude extract are shown in Figure 114. The acidic fraction had the highest specific activity in the absence of S9. The basic fraction, which was only 0.5% by mass of the total, showed enhanced activity in the presence of S9. The neutral fraction showed a somewhat higher mutagenic response in the presence of S9 than in its absence, but the difference was smaller than for the crude extract. It appeared that much of the direct-acting mutagens were in the crude extract and acidic fraction. The neutral fraction, which was 95% of the mass, would be expected to contain most of the mutagenic compounds, including PAH's.

Seven sub-fractions of the neutral fraction were collected by partitioning with organic solvents, and it was found that with the VW Rabbit sample, the percentage of various fractions by mass was fraction 1 -72.9%, fraction 2 - 6%, fraction 3 - 2.1%, fraction 4, 3.8% -fraction 5 - 4.8%, fraction 6 -8.6% and fraction 7 - 1.8%.

Using strains TA98 and TA100, the subfractions were assayed at 6 - 8 concentrations in the range of 2 to 100 ug/plate. The highest direct-acting mutagens were detected in subfractions 3-7 ,with subfraction 4 having the highest specific activity (Figure 115). The activity of subfractions 3-5 were enhanced in the presence of S9, particularly with strain TA100.

The paraffinic subfraction 1 showed no detectable activity and subfraction 2, which contained the PAH's, showed marginal activity only in the presence of S9.

Neutral subfraction 4 and the acid fraction require additional characterization. Subfractions 3, 5 and 6 also need to receive additional analytical attention. The PAH subfraction 2 showed only marginal activity, but many members of this class are recognized as

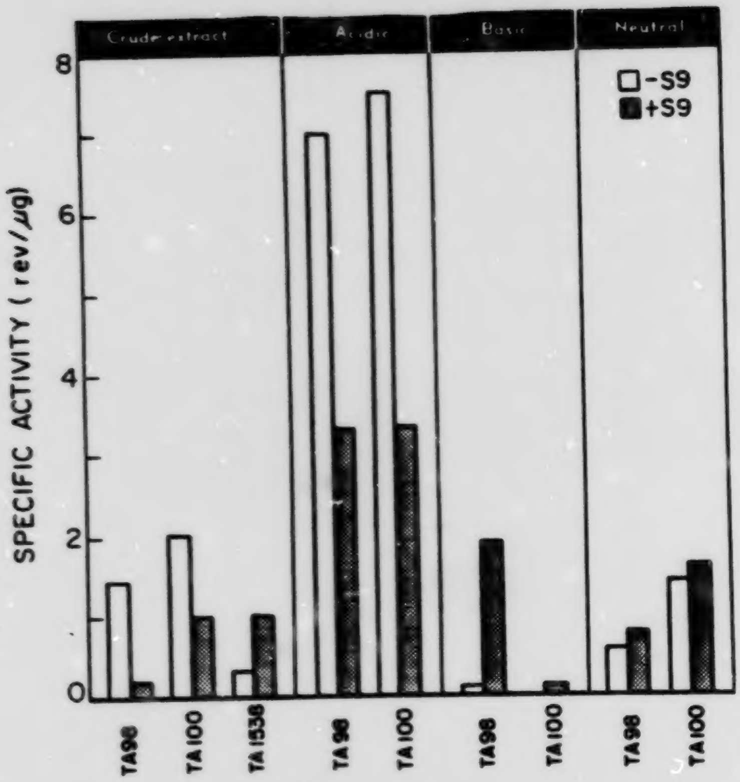


FIGURE 114

Mutagenicity of Diesel Particulate Extract and Its Fractions (VW Rabbit)

(Source: Reference 298)

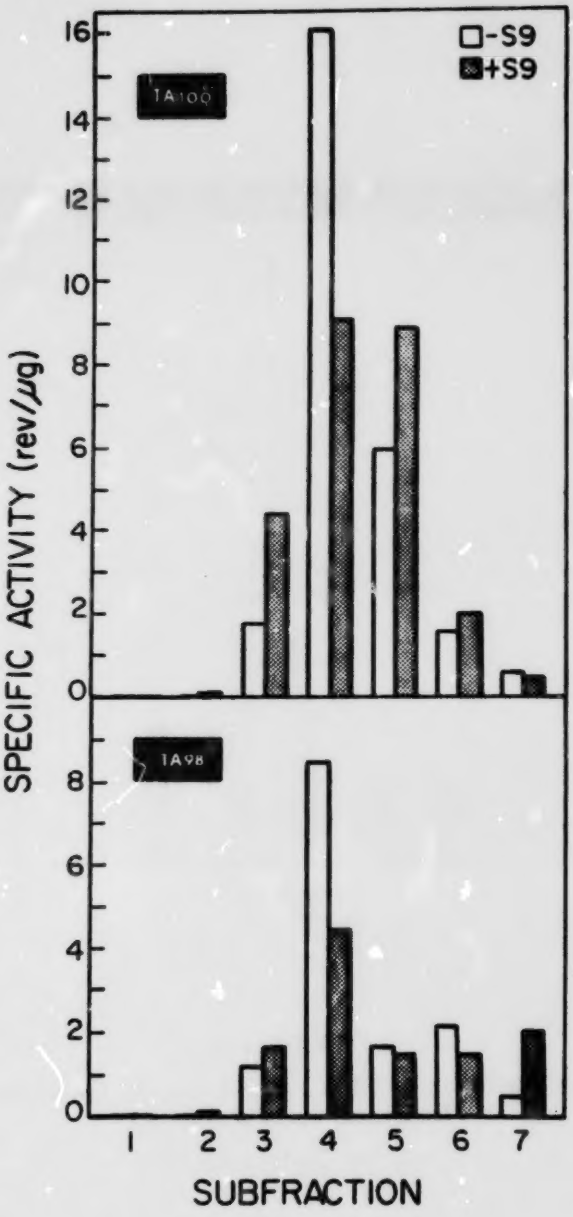


FIGURE 115

Distribution of Mutagenicity Among Subfractions of Neutral Diesel Particulate Extract (VW Rabbit)

(Source: Reference 298)

carcinogens and mutagens. Consequently, the characterization of some of the chemicals in this subfraction need to be delineated. Mass spectrometry indicated a clean separation of the PAH's from the paraffins and other classes of compounds.

Rodriguez et al (299) studied the mutagenic activity of emissions from 2-cycle and 4-cycle diesel engines mounted on dynamometers.

It was found that solvent fractionation of the particulate resulted in a descending order of mutagenic activity. Mutagenicity was concentrated in fractions containing aromatic and/or oxygenated functionalities and seemed to be greater in fractions from the 4-stroke engine. The results using the basic and insoluble tar fractions were ambiguous, partly because of the limited amounts available. Gel chromatography of subfractions provided chromatograms which were more useful than those derived from whole fractions of diesel exhaust. Examples of the chromatograms are shown in Figure 116. The peaks are more discreetly separated, but the "envelope" which results from the incomplete separation of many similar compounds is still present. The same characteristics of earlier chromatograms were again evident, that is, the 2-stroke exhaust was made up of earlier eluting low molecular weight compounds.

The chromatographs allowed spectral enhancement manipulation which resulted in the tentative identification of the compounds listed in Table 109.

It is thought by most investigators that the organic solvent extractable (soluble organic) fraction (SOF) contains the components that are of greatest significance to contamination of the environment (see Section 3.4.7). The SOF has been divided into a number of subfractions and the biological significance of each of these varies, at least in terms of mutagenic potential. The functional groups include acid, base and neutral fractions. The neutral fraction may be further separated by HPLC into nonpolar, moderately polar and highly polar fractions. These are sub-designated consecutively as nonpolar being polynuclear aromatic hydrocarbons, moderately polar as a transition fraction and highly polar fractions as oxygenate fractions, respectively. Because of the popularity of the Ames Test, it has been used to determine the mutagenicity of the SOF. Surprisingly, most of the mutagenic activity in diesel particulate extracts is concentrated in fractions other than the polynuclear aromatic hydrocarbon fraction (PAH). These observations have been made both for diesel exhaust particulate and for ambient air particulate extracts.

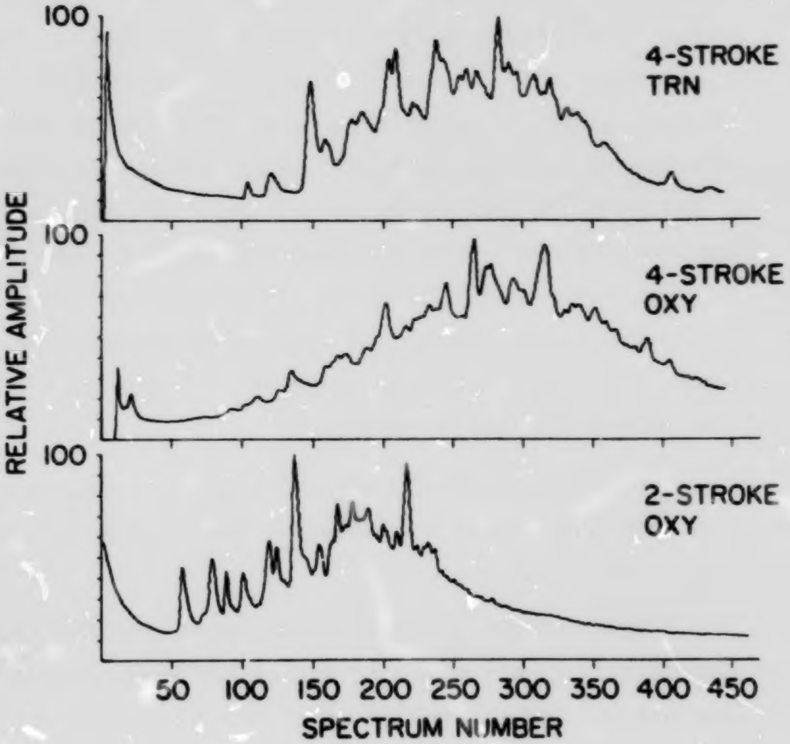


FIGURE 116

Total Ion Chromatograms of Diesel Organic Subfraction 4
(Source: Reference 299)

TABLE 109

alkyl naphthalenes (ARM I, II)
anthracene/phenathrene (ARM I, II)
alkyl anthracene/phenanthrenes (ARM I, II)
fluoranthene (ARM I)
pyrene (ARM I)
benzofluorene and/or methylpyrene (ARM I)
o-phenyl anisole (TRN II)
4-methylphenylbenzo(c)cinnoline (TRN II)
nitropyrene (TRN II)
phenylethylketone (OXY I)
chromone (OXY I)
coumarin (OXY I, II)
formylnaphthalene (OXY I, II)
alkyl naphthols (OXY I, II)
perinaphthindenone (OXY I, II)
anthroquinone (OXY I, II)
benzanthrone (OXY I, II)
m-hydroxybenzaldehyde (OXY I, II)
methoxybenzaldehyde (OXY I)
naphthol (OXY I, II)
p-phenylphenol (OXY I, II)
dibenzo-p-dioxin (OXY I, II)
naphthoic anhydride (OXY I, II)
o-phenylphenol (OXY I)
methylbenzanthrone (OXY II)
anthrone (OXY II)

(Source: Reference 299)

Prior to the studies undertaken by Scheutzle et al (300) and it was believed that the polynuclear aromatic hydrocarbons were primarily responsible for the mutagenic and possible carcinogenic activity of exhaust particulate. For comparative purposes, the authors listed mutagenicity data for solvent extracts of particulate derived from various environmental sources (Table 110).

TABLE 110

Ames Salmonella Typhimurium Mutagenicity for Some Environmental Particulate Solvent Extracts

Source	Mutagenicity (Revertants/mg)
Non-Catalyst Gasoline Engines (Average of Five)	900
Four Cylinder Diesel Engine	2,000
Truck Diesel (4-Stroke)	4,300
Cigarette Smoke Condensate	1,100
New York City (Winter)	1,300
New York City (Summer)	1,600
Residential Area (Japan)	44 - 1,800
Industrial Area (Japan)	290 - 2,200
Power Plant	2,600
Coke Mill	1,200

(Source: Reference 300)

The Ford Motor Research Group of Schuetzle, in cooperation with Harvey and Hunt of the University of Virginia Chemistry Department have reported the identification of more than 100 PAH derivatives in moderately-polar HPLC fractions of diesel particulate extracts. The derivatives included substituents with hydroxy, ketone, quinone, carboxaldehyde, acid anhydride, dihydroxy and nitro groups. Similar derivatives from like fractions have been reported by other investigators (100).

As has been discussed earlier, quantitatively the mutagenicity of most of the identified PAH derivatives is too low to account for the mutagenic activity of total exhaust extracts. This group, however, have demonstrated that nitrated-PAH or nitro-PAH compounds have a significant mutagenic potential and could account for a large portion of the crude extract activity (101, 301). For example, 1-nitropyrene has been shown to be an extremely potent direct-acting mutagen in the Ames assay. Schuetzle et al have found 1-nitropyrene as well as other nitrated PAH's to be present in diesel particulate extracts and to be an important contributor to the mutagenicity of such extracts. Whether the activity resulted from an unusual sensitivity of the test strains of Salmonella to nitro-PAH in being able to convert them to mutagens or whether the nitro-PAH's are formed during the particulate collection and extraction procedures is not known but they consider their presence important in evaluating the implications of bacterial mutagenicity experiments using diesel particulate extracts.

The whole issue of the mutagenicity of diesel particulate extracts is extremely complex. For example, PAH compounds have been shown to react with filter media, nitrogen oxides, sulphur oxides, sulphuric acid aerosols, ozone and smog. For example, a number of additional compounds were identified when smog was drawn over BaP. These included a number of BaP isomers which appear to be dicarbonyl compounds formed by ring-opening oxidations. Some of these isomers, particularly the nitro-BaP derivatives, were found to be direct-acting mutagens.

Several investigators have looked into the identification of PAH derivatives using a number of analytical and instrumental approaches. A number of highly mutagenic and/or carcinogenic compounds have been found, including the alkyl-9-fluorenones and nitro-naphthalene compounds, all of which are highly mutagenic.

The primary objective of Schuetzle et al (100, 101, 300, 301) was to determine the chemical composition of the transition fractions because they appeared to have the most serious effect on the environment and the organisms within the environment. The transition fraction probably represents more than 50% of Ames mutagenicity in the SOF of diesel particulate. Preliminary results were reported on the beta subfraction of the PAH and oxygenate fraction.

Two samples of diesel exhaust particulate were used, but both came from White Beauty diesel engines. The neutral components of the SOF were extensively examined, and were divided into divisions based on resolution and identification using HPLC-MS techniques.

In the nonpolar group of PAH's designated alpha-1 and alpha-2, there were PAH's with 2 - 4 rings and PAH's with 4 -6 rings, respectively. In the beta fraction were found PAH with 6 - 8 rings, hydroxybenzenes and the nitro PAH's. Nitro-PAH's were largely composed of 2 - 6 rings and the hydroxy-PAH's of 3 - 4 rings.

In the moderately polar fraction in what was termed the gamma-1 region, there were PAH-carboxaldehydes with 3-4 rings, PAH quinones of 3 rings and ketones of 2-3 rings. The gamma-2 region contained PAH-quinones with 3-5 rings, hydroxy-PAH with 5-7 rings, PAH-ketones with 3-4 rings, dihydroxy-PAH's with 3-5 rings and dinitro-PAH's consisting of 3 rings.

The highly polar oxygenate group, called the theta-1 fraction, contained PAH-quinones with greater than 6 rings, PAH carboxylic acids with greater than 3 rings and nitrogen-containing heterocyclics. Although there was poor baseline separation between some of these regions resulting in contamination by aliphatics and PAH compounds, making data analysis difficult, considering the complexity of the mixture, the resolution and definition was impressive.

Some of the oxygenated derivatives of PAH were probably formed during storage of the specimens or during the analytical procedures, since they could not have eluted in the beta-fraction. It should be remembered that in the columns some interchange between chemicals takes place, causing even more complex compounds to be formed. The most abundant PAH species in the selective fractions included:

#3-dimethyl (anthracenes or phenanthrenes)
#5-cyclopentapyrene
#8-diphenylbenzene
#9-benzo(a)anthracene + isomers
#11-methyl diphenylbenzene
#12-methyl benzo(a)anthracene
#14-dihydro (benzo(a)pyrene + isomers)

When investigating the gamma-1 and gamma-2 fractions, which contained the oxy-PAH's of potential mutagenic concern, it may be that some species, such as aliphatic hydrocarbons and phthalate esters, were present as contaminants derived during HPLC fractionation and concentration procedures.

The nitro-PAH's were difficult to analyze by gas chromatography/mass spectrometry (GC/MS), largely due to their high reactivity and undoubtedly there was some loss of the species during analysis. Additional definition of the nitro-PAH's was carried out using high resolution mass spectrometric (HRMS) and GC/MS techniques.

The mass recovery of the diesel extract injected into the HPLC was greater than 96%. One hundred and three (103) percent of the direct-acting and 91% of the indirect-acting mutagenic activity injected into the HPLC was recovered.

Although the PAH's required enzymatic (S9) activation in order to attain a mutagenic response, many of the nitrated oxygenated-PAH's were direct-acting mutagens. Nitrated and oxygenated PAH species were not detected in the alpha-1 or alpha-2 fractions which correlates with their not having direct-acting mutagenicity.

The beta fraction of the nonpolar-PAH group did show significant levels of direct-acting mutagenicity. This may have been as a result of incomplete separation from other fractions.

The largest proportion of direct-and indirect-acting mutagenicity was contained in the moderately polar and neutral transition groups, in particular the gamma-1 fraction. Many aliphatic hydrocarbons and phthalate ester contaminants were present which were difficult to eliminate because of their ubiquitous nature.

The most abundant of the group were the oxy-PAH species which amounted to 39% of the total identified components, as well as ketone and hydroxy PAH derivatives containing 3 - 4 rings. A significant component was 9-fluorenone, but this did not show direct or indirect mutagenicity. Because of separation difficulties, a quantitative estimate of the hydroxy and PAH-ketone species was not possible.

In this subgroup, the third most abundant species was the carboxaldehyde PAH derivatives. The only bioassay data reported in the literature is for 6,4-benzo(a)pyrene carboxaldehyde, which has been shown to induce tumours in mice.

Minor components include nitro-compounds and 1-nitropyrene was positively identified in an early eluting subfraction of the gamma-1 fraction.

These nitro-PAH compounds represented a significant portion of the mutagenic activity of the fraction.

The gamma-2 fraction had direct-acting mutagenic activity representing 9 -15% of the total extract activity. This fraction, however, was largely made up of oxygenated-PAH species. Within this group, the most abundant were the anhydrides of naphthalic and methyl-naphthalic acid. Other acid anhydrides were also present in this fraction (Table 111).

The theta-1 fraction was primarily comprised of carboxylic and dicarboxylic acids with some lesser oxygenated and multiple cyclic-PAH's.

It is not known whether the oxy-PAH derivatives observed in the study were formed during the combustion process, in the exhaust system or during sample collection.

Mutagenicity data for the PAH species identified in this study were not available. However, as mentioned earlier, 1-nitropyrene was estimated to comprise the largest part of the gamma-1 fraction. With

TABLE 111

Ames Salmonella typhimurium Mutagenicity For Some PAH and Nitrogen and Oxygen Containing Derivatives of PAH

	Activity (revertants/mg)	
Compound	TA98 -S9	TA98 +S9
N and O-PAH		
Benzo(e)pyrene-9, 10 Epoxide	--	2,700,000 (52)
1-Nitropyrene	1,400,000 (60)	--
Nitrobenzo(a)pyrene (1 + 3 Isomers)	707,000 (59)	17,500,000 (59)
4,5-Dihydrodihydroxy Benzo(e)pyrene	--	480,000 (52)
2,4,7-Trinitrofluorenone	95,000 (57)	--
2,7-Dinitrofluorenone	75,000 (57)	--
Benzo(e)pyrene-4,5 Dihydrodiol	--	53,600 (52)
2,7-Dinitrofluorene	35,000 (57)	--
2-Nitrofluorene	34,000 (23)	--
Acetylamino Fluorene	17,500 (57)	--
Pyrene Oxidation Products	8,400 (59)	--
Hydroxy Phenanthrene	7,100* (56)	--
9,10 Phenanthrene Quinone	7,100* (56)	--
Benzo(a)pyrene Oxidation Products	7,200 (59)	--
1,5-Dinitro-2 Methyl-9,10 Antraquinone	280* (47)	5,200* (47)
Trihydroxy Anthraquinone (Anthralin)	1,700 (57)	--
Cyclopenteno (c,d) pyrene Anhydride	300 (26,40)	--
Benzanthrone Oxidation Products	219 (59)	--
9,10-Dihydroxy Anthracene	Toxic (56)	Toxic (56)
9,10-Anthracene Quinone	Toxic (56)	Toxic (56)
PAH		
9,10 Dihydro Benzo(e)pyrene	--	278,000 (52)
1,9-Dimethyl Fluorene	--	90,000* (54)
Benzo(a)pyrene	--	45,000 (54)
10-Methyl Benzo(a)pyrene	--	33,000 (54)
Benz(a)anthracene Fraction - Syncrude	--	6,600 (53)
PAH + ME - PAH Fraction - Syncrude	--	3,800 (53)
9-Methyl Fluorene	--	2,500* (54)
Multialkylated PAH Fraction - Syncrude	--	2,220 (53)

(*) TA100 used instead of TA98.

(Source: Reference 300)

this in mind, it was estimated that the 1-nitropyrene accounted for 44% of the gamma-1 fraction mutagenicity or 30% of the total extract activity.

The nitro-PAH's probably accounted for a significant portion of the direct-acting mutagenicity of this fraction. However, exact designation of this activity awaits better bioassay and chemical quantitation of nitro-PAH.

The same group (Salmeen and co-workers (301) reported the results of additional work which found that 1-nitropyrene constituted a significant portion of the mutagenic activity of exhaust extracts collected from passenger vehicle diesel engines, with the percent contribution varying from 1 to 24%. They suggested that even a 1% contribution was important when considering the thousands of compounds present in the particulate extracts. Overall, the data suggest the possibility that a small number of compounds may account for a large portion of the mutagenic activity of the complex exhaust mixtures.

Pederson and Siak (302), on the other hand, feel that diesel extract mutagenic activity is associated with components that are more polar than 1-nitropyrene or other mono-substituted nitro-PAH's.

Henderson et al (303) came to a similar conclusion to Pederson and Siak. Using a sophisticated mass-spectrometer technique for analysis, he found that the even-mass ions or dinitro-PAH's appeared to correlate with increased mutagenic activity in diesel particulate extracts, while odd-mass ions (mononitro-PAH's) were negatively correlated with mutagenic activity. The even-mass ions of particular significance were tentatively identified as dinitrophenanthrenes, dinitromethylbiphenyls and dinitronaphthalenes containing 2, 3 and 4-methyl groups. It was suggested that these compounds might be particularly important in the total mutagenicity of diesel soot extracts. Nitropyrenes did not significantly correlate with differences in the mutagenicity of various diesel exhaust samples, although the compounds possibly accounted for 5 - 10% of the total mutagenicity.

Mutagenic compounds were separated using TLC and HPLC techniques and mutagenicity was tested in the Ames Salmonella mutation assay. About 1/3 of the mutagenic activity in the particulate extract was found in the fractions containing the mono-substituted nitro-PAH compounds. Chemical characterization suggested that 1-nitropyrene was the predominant nitro-PAH present in these fractions. However, further investigation indicated that the amount of 1-nitropyrene present would account for less than 15% of the mutagenic activity, pointing to the presence of other mutagens, probably nitroarenes present in the nitro-aromatic fraction which were more mutagenic than 1-nitropyrene. It was suggested that the dinitropyrenes might account for 15 - 20% of the mutagenic activity in the particle extracts (302).

Pederson and Siak (302) also examined whether mammalian enzyme systems could activate the chemical compounds in particle extracts to mutagenic products as readily as did bacterial enzymes. The enzymatic steps in the bacterial activation of nitro-aromatic mutagens involved a reductive process in which the first step is probably a one electron reduction to a nitro-aromatic anion-free radical followed by additional reductions to form amines, but the intermediates responsible for the mutagenic activity are not known. Under appropriate conditions, the activation of mutagens in diesel particle extracts by rat liver S-9 enzymes was demonstrated as a difference between assays with and without NADPH. One-nitropyrene was also activated by S-9 enzyme activity. The results showed that NADPH-dependent enzymes in the rat liver S-9 fraction increased the mutagenic activity of both diesel particulate extracts and 1-nitropyrene in bacterial mutation assays. Although the study showed that rat liver enzyme systems could convert components of diesel extracts into products which were mutagenic in bacterial systems, two important reservations were expressed by the authors in extrapolating the significance of these findings to the in vivo situation in man. The first was that the use of a liver homogenate as an enzyme source was a crude and limited approximation of the activity found in intact cells and tissues. The second was that the target origin was the lung, not the liver, and the enzymatic processes in the lung might be significantly different from those in the liver.

King, Tejada and Lewtas (304) attempted to evaluate the removal and release of mutagenic activity and 1-nitropyrene from diesel particulate in the presence of rabbit alveolar macrophage cells obtained by lung lavage. After incubation, the harvested macrophages were separated and sonicated to release engulfed particles and extracted with dichloromethane and methanol. Extracts of the medium, free particles, engulfed particles, cell sonicate and water washes were subjected to Ames testing. It was found that the presence of macrophages decreased the mutagenicity of diesel particulate by 62%. As well, significantly less 1-nitropyrene was recovered from the macrophage-treated group (76%). Since over 95% of the particles were phagocytized and only 27% of the original 1-nitropyrene was recovered from the engulfed particles, it appeared that the macrophages metabolized the 1-nitropyrene to a non-detectable form. A comparison of the recovery showed a greater loss of mutagenicity than 1-nitropyrene suggesting that other compounds in the particles may have contributed more to the mutagenicity than the 1-nitropyrene alone.

Kohan and Claxton (305) also concluded that 1-nitropyrene may not be the major mutagenic component extractable from specific diesel particulate samples.

Others (306) have also questioned which of the PAH's are the most carcinogenic and have suggested that nitroarenes may be significant.

Nitrogen dioxide and nitric acid have been shown to react with polycyclic aromatic hydrocarbons, forming nitroarenes (Table 112) which are mutagenic in the Ames test (see also section 3.4.4). It is not known if such reactions occur in ambient air or on the filter at the time of particulate collection. Some nitro compounds were shown to have the property of becoming more mutagenic when incubated with Salmonella TA98 under anaerobic conditions. This property was used to investigate the presence of similarly behaving nitro compounds in environmental samples (307). Urban particulate matter collected in Stockholm had an enhanced mutagenicity under anaerobic assay (Table 113), but periods with little enhancement have also been recorded,

TABLE 112

Mutagenicity in the Absence of the Microsomal System of Some Nitroarenes in TA 100 and TA 98 Under Aerobic (Normal) and Anaerobic Conditions. The Mutagenicity is Given as Revertants/ug of the Commercial Sample Assayed Without Purification

	TA 100	TA 98	
	Aerobic	Aerobic	Anaerobic
1-nitronaphthalene	15	1.0	2.2
2-nitronaphthalene	24	2.5	5.8
9-nitroanthracene	1	<1	63*
3-nitropyrene	400	3,700	8,200

(*) this increase is caused by the major component of the sample having a retention as expected for nitroanthracene.

(Source: Reference 307)

TABLE 113

Relative Mutation Frequency of Exhaust Samples and Urban Particulate Matter in TA 100 and in TA 98 Under Aerobic and Anaerobic Conditions. The Mutagenicity is Expressed as Percent of the Response in TA 98 -S-9 Under Normal Conditions. The Samples are the Same as Those Reported in Table 112

	TA 100	TA 98	
	Aerobic	Aerobic	Anaerobic
GASOLINE			
particulate matter	200	100	130
condensate	140	100	120
DIESEL			
particulate matter	180	100	130
condensate	150	100	90
URBAN PARTICULATE MATTER	95	100	220

(Source: Reference 307)

reflecting the difference in the composition of air pollutants. Anaerobic incubation of diesel and gasoline exhaust samples gave little or no increase in mutagenicity indicating that mutagenic nitro compounds causing an enhanced mutagenic effect are less abundant in exhaust samples than in most samples of urban particulate matter. Undoubtedly there exists mutagenic nitro compounds which do not cause an enhanced mutagenicity under anaerobic assay conditions. However, further investigation is needed to define these.

Extraction of mutagenic compounds from gasoline engine crankcase oil with DMSO gave the highest recovery among several procedures tested. Fresh oils had no detectable mutagenicity, whereas used oil extracts had activity. Used crankcase oils have compounds which are both mutagenic in the absence of S9 as well as compounds that have increased mutagenicity with S9. The mutagenic property of motor oil increased with age of use (see Figure 117). Attempts to assay motor oils from diesel engines were less successful. Extracts of such oils caused a small increase in the reversion frequency, but the effect was not dose-dependent which prevented an assessment of the mutagenic potency.

The presence of mutagenic compounds in used crankcase oil has been reported before, as has the presence of mutagens requiring mammalian metabolic activation. Extraction procedures may not extract all mutagenic components, but it favourably separated mutagenic compounds from components which were toxic to, or interfered with the assay. It should be noted that the failure to extract readily-detectable amounts of mutagenic components in used motor oils from diesel engines may have been due to their content of mutagens, which may be low, or it may be due to the fact that the partition coefficients were unfavourable. Mixing used motor oil from a diesel engine with an equal amount of used motor oil from a gasoline engine gave, in the extraction, the same mutagenicity as when extracted from gasoline engine oil. This demonstrated that toxic or interfering components from diesel engine motor oil probably are not causing a false low mutagenicity of the diesel engine motor oil extracts.

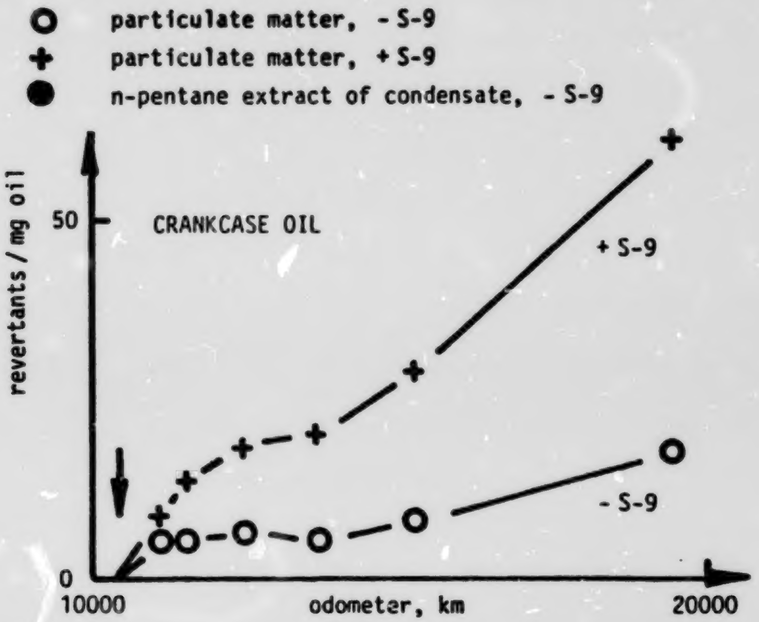


FIGURE 117

Mutagenicity in TA 98 +/- S-9 of Motor Oil From a Gasoline Powered Car. Samples were Collected at Various Times Between Services Over a Period of 140 Days. Fresh, Non-mutagenic, <2 revertants/mg, Oil was Used from ↓ .

(Source: Reference 307)

One study (307) indicated an unspecified high mutagenicity of particulate material from diesel engines, probably direct-injection engines. Consequently, a comparison between diesel and gasoline exhaust was made. An average mutagenicity of 100 revertants per litre of exhaust was derived for diesel samples and 15 revertants/l of exhaust from gasoline sources (Table 114). Exhausts are known to contain mutagenic and cancerogenic PAH's, such as BaP, which require mammalian metabolism for the formation of mutagenic metabolites. Their detection in a complex mixture by the Ames test depends on their concentration relative to the concentration of components not requiring mammalian activation. The present investigation showed only particulate matter from gasoline exhaust had a composition which favours this detection. Gasoline exhaust, has however, a considerable S9 dependent mutagenicity residing in the basic fraction.

TABLE 114

	Diesel	Gasoline
Revertants/liter exhaust	100	15
Revertants/g fuel consumed	2,700	190
Revertants/test cycle (12.1 km)	2,200,000	180,000

(Source: Reference 307)

The study underscored the importance of collecting condensable gaseous components which are present in the condensate. It seems likely that such components, given sufficient time, may become adsorbed to particulate matter in ambient air.

Fractionation with respect to polarity (Table 115) showed that the distribution of components was not the same for different samples. There was a wide distribution of mutagenicity amongst several fractions, which implies that no single compound or single type of compound alone is responsible for a major part of the mutagenesis of the sample.

Risby et al (308) attempted to identify the compounds, or groups of compounds, with biological activity and relate this to engine operating conditions and fuels. A pure hydrocarbon fuel and a low ash synthetic lubricating oil were used in place of a full boiling-range diesel fuel and a traditional lubricating oil. The use of synthetic low-ash lubricating oil minimized the contribution made by any partially combusted lubricating oil. Also, the engine, a light-diesel engine (350 Oldsmobile) cradled in a dynanometer, was run under constant operating conditions.

TABLE 115

Relative Distribution of Mutagenicity in Different Fractions As Assayed with the Strain TA 98, Expressed as Percent of the Mutagenicity of the Unfractionated Sample. Exhaust Samples Used for Fractionation Were Obtained by Combining Extracts from Several Test Cycles

	Diesel Exhaust 9016-19		Gasoline Exhaust 8903-07+8912-16				
	Particulate Matter	Condensate[a]	Particulate Matter		Condensate[a]	Urban Particulate Matter	
Fraction	-S-9	-S-9	+S-9[b]	-S-9	-S-9	+S-9	-S-9
Basic	2	≤4	3	≤7	<2	4	4
Acidic	7	8	4	6	5	13	19
Aliphatic	15	6	<2	<3	14	<2	<2
Aromatic	21	15	32	8	35	15	8
Oxygenated-I	13	3	35	21	10	28	19
Oxygenated-II	--[c]	--	--	--	--	3	4
Sum of Fractions	58	32	104	29	64	63	54
Reconstituted Sample	--	--	--	--	--	62	60

(a) pentane extract
(b) 20 ul S-9 per plate
(c) not investigated

(Source: Reference 307)

The simple hydrocarbon fuels employed produced exhaust particulate matter which had biological activity which was accounted for by reaction mechanisms found under the engine combustion conditions used. Biological activity was found in the total extract, but raw particulate matter did not exhibit the same properties. The significance of this could be that compounds adsorbed onto particles are not readily released for toxicological effect under physiological conditions and consequently may not represent a health hazard.

The use of filters to collect particulate matter may lead to particulate chemical reactions as a result of volatile reactive species. In one study, benzo(a)pyrene reacted with nitrogen dioxide to produce mono-nitro-benzo(a)pyrene, which has mutagenic activity without activation. Thus, one might conclude that compounds undergo similar reactions with oxides of nitrogen which are present in diesel exhaust emissions. This concept may be significant when assessing public health problems. The biological activity of extracts from particulate matter of the pure hydrocarbon fuel and the full boiling range fuel did not differ significantly. Consequently, it was suggested that bacterial activities, which were observed for extracts from particulate matter from diesel engine exhaust, were the result of combustion and not artifacts from the fuel itself.

At a recent symposium, Zweidinger (4, 284) compared the emission characteristics for a light-duty diesel and several heavy-duty truck diesel engines and provided data as to the relative mutagenic activity of their emissions. Total hydrocarbon, carbon monoxide, NO_X, particulate and SOF were all higher for the heavy-duty diesels than for the light-duty engine. However, the mutagenic activity in the emissions from all vehicles were of similar orders of magnitude. A notable difference between the heavy and light diesel engines was the lower BaP emissions from the heavy-duty engines (Tables 116, 117, 118 and 119).

TABLE 116

FTP Emissions for In-Use Light Duty Gasoline and Diesel Vehicles

Emission Factors	Unleaded Gas (15)[A]	Diesel (6)
THC, g/mi	1.05[B] (0.22 - 2.94)[C]	0.38 (0.21 - 0.60)
CO, g/mi	12.2 (1.6 - 28.0)	1.27 (1.03 - 1.86)
NO_X, g/mi	2.35 (0.2 - 6.1)	1.27 (0.8 - 1.9)
Particulates, mg/mi	21.0 (5.9 - 36.1)	607 (370 - 1070)
SOF, mg/mi	4.9 (0.7 - 13.5)	124 (45 - 290)

A - Number of Vehicles
B - Mean Value
C - Minimum and Maximum Values Observed

(Source: Reference 4)

TABLE 117

FTP Particulate Emissions for In-Use Light Duty Gasoline and Diesel Vehicles

Emission Factors	Unleaded Gas (15)[A]	Diesel (6)
BaP, ug/mi	2.1 (0.1 - 12.4)	4.5[B] (0.9 - 7.7)
1-Nitropyrene, ug/mi	0.19 (0.004 - 1.21)	7.8[B] (3.4 - 10.5)
TA98 -S9, Rev/ug	8.0 (0.4 - 19.6)	4.1
TA98 +S9, Rev/ug	14.2 (1.3 - 42.2)	---
TA98 -S9, Rev/mi[C]	40.6 (2.3 - 181)	509 (260 - 670)
TA98 +S9, Rev/mi[C]	71.0 (3.3 - 424)	---

A - Number of Vehicles
B - Typical Values
C - x 10^3

(Source: Reference 4)

TABLE 118

HDV Truck Transient Emission Characteristics

	Gasoline		Diesel			
	Ford 370	HI 345	Cat. 320B	Mack 676	Cumm. 290	DD 8V71
GVW Lbs. (x 10-3)	19.7	24.0	27.5	80.0	80.0	36.9
HC, g/mi	20.9	6.2	1.90	1.43	2.32	2.13
CO, g/mi	129	103	5.1	9.5	8.0	75.1
NO_X, g/mi	13.0	13.7	19.3	29.6	27.4	35.5
Particulate g/mi	0.58	0.89	0.90	1.95	1.61	3.33
SOF, mg/mi	40.6	14.5	475	193	336	537

(Source: Reference 4)

TABLE 119

HDV Truck Transient Emission Characteristics

	Gasoline		Diesel			
	Ford 370	HI 345	Cat. 320B	Mack 676	Cumm. 290	DD 8V71
BaP, ug/mi	61.0	17.1	1.61	0.92	5.30	1.33
TA98 -S9 Rev/ug	2.57	8.05	1.10	1.36	1.02	0.04
TA98 +S9 Rev/ug	14.9	17.4	1.08	1.22	0.95	0.09
TA98 -S9 Rev/mi^A	104	117	523	262	343	21.5
TA98 +S9 Rev/mi^A	604	253	515	232	319	48.3

A - x 10^3

(Source: Reference 4)

The author also provided comparative data for gasoline engines and noted that gasoline vehicles had a higher Ames test activity with activation while the reverse was true for diesel engines. It must be remembered that even the heavy-duty engines were above ground vehicles which would not likely be operated at the same fuel:air ratio as engines used underground in mines, and thus the emissions could be distinctly different.

Other groups have published additional data, which will not be discussed in detail, relating to the mutagenicity of diesel exhaust particulate and extract components (309).

Pierson and co-workers from the Ford Motor Company, in cooperation with the U.S. EPA, reported the results of experiments to evaluate the mutagenic and general chemical properties of diesel particulate organic matter in a realistic near-road environment conducted in the Alleghany Mountain tunnels of the Pennsylvania Turnpike in 1979 (310). Samples were collected at various times when either diesel traffic or gasoline powered vehicles dominated the roadway. A detailed report of their findings is available, but in summary, they found that the results were indistinguishable from dynamometer/dilution-tube results in terms of diesel particulate chemical properties such as mass emission rate, extractability into methylene chloride, particle size distribution and extractable mutagenicity. Highway traffic produced a readily observable increase in the mutagenicity of the atmosphere near the road. Per distance driven, the mutagenicity of the extractable particulate emissions from heavy-duty diesels was many times those of light duty spark-ignition systems. It was suggested that the dynanometer/dilution-tube method is probably a valid index of the on-road situation.

Of peripheral interest is the work of Dehnen and co-workers (311) who pointed out that although the mutagenic activity of atmospheric particulate matter had been attributed to the presence of PAH, samples of air in which no BaP could be detected, also proved to be highly active. To further investigate this point, they measured the mutagenic activity, using the Ames test, of particulate extracts and fractions of atmospheric particulate, giving special attention to the

PAH fraction. Sampling took place in a German iron and steel production town and the particulate matter was extracted with methanol, fractionated using a liquid-liquid partition system into a PAH fraction and a fraction retained on an alumina column, and eluted with propanol-2. This latter fraction contained, among a large number of unknown components, aza-heterocyclic compounds. In agreement with the results of other authors, the whole extract proved to have mutagenic activity. The correlation between the PAH content and mutagenic effect was not unequivocal and it was thought that samples from different sites contained different pollutants which accounted for varying degrees of mutagenicity. It was concluded that other substances besides PAH participate in the mutagenicity of airborne pollutants. A considerable proportion of the mutagenic activity did not require metabolic activation.

Moller and Alfheim (312) also found airborne particulate matter to be mutagenic and to contain mutagens other than PAH which require metabolic activation by the addition of liver microsomes.

Wei, Wang and Rappaport (281) published a commentary on the significance of the Ames Test in determining the carcinogenicity of diesel emissions. They pointed out that the data available show that diesel exhaust samples collected by filtration or electrostatic precipitation are mutagenic when assayed in the Ames Test. They, however, also noted that the mutagenic potency of a "standard" diesel exhaust sample was difficult, if not impossible, to state in absolute terms relative to the potency of other know mutagens. Diesel exhaust samples generally behave as direct-acting mutagens which is an important point in that BaP and the basic mutagens in tobacco tar are indirect-acting mutagens. It was suggested that the principle mutagens in diesel exhaust must therefore be different from the mutagens in coke oven emissions or tobacco tar. In fact, the addition of S-9 liver supernatant, which activates indirect mutagens, decreased the mutagenicity of diesel exhaust samples. The authors posed the question as to the chemical identity of the direct-acting mutagens in diesel exhaust products. They suggested that the unsubstituted PAH's, which occur at low concentration on diesel particulates, could not account for the liver-independent mutagenic activity of the samples.

Some of the nitro compounds might merit consideration, as well as the polycyclic aromatic hydrocarbons, especially those with multiple rings which can readily react with dilute nitrous or nitric acid to form nitro-derivatives. They were unable to clearly define which components might be the direct-acting mutagens and emphasized that mutagenicity in the Ames test was not sufficient evidence to imply a hazard to human health.

A review of the specific Ames activity of whole extracts of diesel exhaust particulate for various engines was compared by Mogan (314) to actual underground and dynamometer-derived data for a Deutz engine (Figure 118). The underground Ames test values were surprisingly similar to those obtained at the ORF using a dynamometer-mounted Deutz engine. These values were also quite similar to those reported for other heavy and light duty diesel engines.

FIGURE 118

Engine	**Olds 350**	**Heavy Duty**	**Heavy Duty**	**CAT 3304**	**CAT 3208**
Source	NYS, EPA	Ford (Alleghany Tunnel)	Berkley	EPA	MTU (Mode 9&10)
REV/ug TA98-S9	0.4 to 4.1	0.4 ± 0.1 to 1.1 ± 0.6	0.46 to 0.8	0.3	0.61 to 0.74
REV/ug TA98+S9	1.1 to 1.6	0.3 ± 0.04 to 0.9 ± 0.2		1.6	

Engine	**CAT 3208 + PTX**	**Deutz 6L714**	**Deutz 6L714 + PTX**	**Deutz 6L714**	**Deutz 6L714 + PTX**
Source	MTU (Mode 9&10)	ORF 16:1 (rated speed 7/8 Load)	ORF 16:1 (rated speed 7/8 Load)	Laurentian (LHD, INCO)	Laurentian (LHD, INCO)
REV/ug TA98-S9	1.3 to 2.2	0.34	3.9	0.35 & 5.6*	2.7 & 4.0
REV/ug TA98+S9		1.5	2.1	0.54 & 0.99	1.1 & 1.3

* Contaminated with Blasting Fume?

(Source: Reference 314)

4.5.3 Mutagenicity of Diesel Exhaust Using Insect and Mammalian Cell Systems

The mutagenic potential of chemicals found in diesel exhaust emissions is influenced by pharmacokinetic and metabolic factors that can only be evaluated in in vivo studies. There are probably synergistic effects on biotransformation pathways as well as interactions at membrane and intracellular sites of target organs important to the ultimate expression of the effects of diesel exhaust in animal systems.

As with the Ames bacterial mutagenicity test systems, mutagenic responses as evaluated in mammalian cell systems are complex. For example, a mutagenic response to BaP has been detected in the mouse lymphoma assay only when activated (315). Of the diesel engine emission samples tested, emissions from a Nissen diesel engine were cytotoxic. Of several samples investigated, those from gasoline engines caused the highest increase in mutation frequency both, with and without metabolic activation.

There was an absence of recombinogenic responses to diesel and other related environmental emission samples, such as cigarette condensates, in a yeast assay. This may have been related to the short exposure times or to the inability of the components to cross the yeast cell wall.

Both the in vitro lymphoma cell mutagenesis and the sister chromatid assay tests are considered mammalian assays in which mutagenicity and/or DNA-damaging capabilities of PAH's such as BaP are detected only following metabolic activation. The failure of the sister chromatid test to detect DNA-damaging effects of cigarette smoke condensate and heavy-duty Caterpillar diesel engine emissions without activation may have been related to the relatively short exposure time used for the sister chromatid testing with activation.

Increased induced mutation frequency in lymphoma cells following exposure to all of the emission samples in the absence of activation, as well as the increases in the sister chromatid frequencies in Chinese hamster ovary cells following exposure to all samples except the Oldsmobile diesel emission would indicate that, in contrast to benzo(a)pyrene, the emission samples contained one or more substances that were direct-acting mutagens and chromosome-damaging agents.

Interestingly, the Chinese Hamster ovary cell (CHO) is not only a useful tool in determining mutagenicity of compounds, but it also mimicks the response of macrophages when exposed to particulate matter (335). Garrett et al (335) found that both rabbit alveolar macrophages (RAM) and CHO were capable of phagocytizing a variety of particulate matter including silica and fly ash from coal combustion. It had previously been shown that the cytotoxicity of silica particles was due to the release of hydrolases into the cell after damaging lysosomal membranes. These investigators showed that cell viability and ATP in the short-term RAM assay and cell cloning in the CHO assay were good indicators of the cytotoxicity of silica. Uncoated fly ash, however, was relatively non-toxic in the RAM assay when determined by measurement of cell number and viability, ATP and protein. The CHO clonal assay was more sensitive as a test for the toxicity of fly ash and the smaller particles were found to be the most toxic.

Pereira (316), using a sperm morphology bioassay found various abnormalities in sperm shape, both in control and diesel-exhaust treated animals. However, the percentage of abnormal sperm significantly increased in animals exposed to the diesel exhaust emissions. The frequency of sperm abnormalities varied among different inbred strains, but it was relatively constant with a particular animal strain or species. Thus, the difference in the frequency of abnormal sperm could be significant, even when small differences occur.

Using a micronucleus bioassay, some increase in the frequency of micronuclei was observed in animals exposed to diesel exhaust for 6 months. By applying the students "t" test to the data, they demonstrated a significant increase in micronuclei in hamsters exposed to diesel exhaust, but in mice exposed to diesel exhaust for only two weeks, there did not appear to be an increase of micronuclei.

A chromosomal aberration bioassay indicated that in both control and diesel-treated animals, the number of polyploid cells were the same. There was no aberration in either chromosomes or chromatids in either the controls or the treated animals. It was apparent that cytological abnormalities were not caused by diesel exhaust.

By administering certain carcinogens, such as 7,12-dimethylbenz(a)anthracene within 24 hours of a partial hepatectomy liver cancer can be developed. Using this bioassay, the carcinogenicity of diesel exhaust was examined (316).

After 3 - 6 months exposure, no significant numbers of foci of gamma glutamyl transpeptide-A hepatocytes developed in the treated groups of rats. It was remarkable that there was no liver toxicity after 6 months of exposure to the particulate and its carcinogens. It was evident that the bioassay used in the study was unable to reveal the presence of either carcinogens or hepatotoxins in diesel exhaust. This could be due to sequestration of carcinogens and hepatotoxins by exhaust particulate so that they were not distributed systemically or the rat liver foci bioassay, as employed in these studies, was not sufficiently sensitive.

Guerrero and co-workers (317) used four to six week old Syrian hamsters to clean air or to air which contained diesel exhaust particulate at concentrations up to 6.39 mg/m^3 ± 0.78 mg/m^3 for 8 hours a day, 7 days a week for a period of about 3 months. Using values obtained in this study it was calculated that the animals would accumulate a particulate load of 15.6 mg. However, losses due to the particulate impacting on the nasal mucosa, moving out on the mucociliary clearance system and through phagocytosis by the pulmonary macrophages, was estimated to reduce the particulate load to 1.5 - 3 mg/animal. The dose-response data from this study suggested that no significant increase in sister chromatid exchange (SCE) would be expected for this level of diesel particulate exposure. It is reasonable to assume that the SCE test, which is extremely sensitive, shows measurable response following 3 months of chronic exposure. Pulmonary tumour production was highly unlikely. Since SCE increases were induced with intratracheal instillation of particulate, it was necessary to assume that longer exposures and higher concentrations of particulate would show a statistically significant increase in the sister chromatid exchange response.

An attempt was made to determine which genotoxic agents of diesel exhaust had activity assuming they must be deposited in the lungs, eluted from the particles and distributed throughout the body (4, 318).

Micronuclei result when there is encapsulation by the membrane of chromosomal material that did not segregate with the spindle apparatus as a result of chromosome breakage or disruption of the single spindle apparatus. This investigation reports an attempt to detect the absorption and systemic distribution of genotoxic agents in diesel exhaust by: 1) metaphase analysis, 2) micronuclei and 3) urinary mutagenicity.

Metaphase analysis of the 7-week diesel exhaust exposures and controls found no indication of chromosomal or chromatid damage. Benzo(a)pyrene at the low dose (1 umole/kg for 4 days) used was inactive. This dose of BaP was chosen to approximate the dose of BaP expected to result from the diesel engine exhaust exposure.

The micronuclei bioassay was performed on mice and hamsters exposed for one, three and 7 weeks to diesel exhaust from a Nissan 6-cylinder engine at a particulate load of 12 mg/m^3, 8 hours per day, 7 days a week. There was no significant difference in the number of micronuclei in polychromatic erythrocytes or red blood cells between controls and mice exposed to diesel exhaust. Benzo(a)pyrene was also inactive.

Urine samples were collected at 1, 3 and 7 weeks in the same mice exposed to either clean air or diesel exhaust. Again, the cyclophosphamide and not the benzo(a)pyrene detected in both the unconcentrated and concentrated samples with strains TA1535 and TA100.

The metaphase analysis of the micronuclei assay detects chemicals capable of breaking chromosomes and chromatids and disrupting the spindle apparatus. The urinary mutagenicity assay will show the presence of chemical mutagens. The combined protocol used in this study was unable to detect a systemic distribution of genotoxic agents resulting from diesel exhaust emissions.

The failure to detect these genotoxic agents could have resulted first from the level of the toxic agents in diesel exhaust being too low to be detected, or secondly, the route of exposure which was inhalation, and finally the absorption of the genotoxic agents onto particles which did not allow them to be absorbed, distributed and metabolized. The combined protocol was not sensitive to benzo(a)pyrene at 1 umol/kg for 4 days.

As had been noted by others, Li and Royer (319) found that extracts of diesel exhaust particulate were cytotoxic, but not mutagenic, to mammalian cells. The low mutagenicity of diesel extracts towards mammalian cells was considered to be due to the reaction of the active compound with cellular components which thus protected the genetic material. It was postulated that before the reactive species could reach the cellular DNA to induce a mutation, many of the other cellular components reacted with the diesel extract thus resulting in a low probability of surviving and expressing the mutation. The lower mutagenicity of the extract in mammalian cells may also be a function of specific bacterial metabolic systems which are absent in mammalian, as compared to bacterial cells. The decrease in cytotoxicity of the diesel exhaust particle extract by serum, bovine serum albumin and sulphhydro compounds etc., suggested that the toxicity of the extract could be easily reversed. Whether this occurs in vivo or is of toxicological significance is not known.

In a later report, Li and co-workers (320) found that DMSO extracts of automobile diesel exhaust had low mutagenic activity in Chinese hamster ovary (CHO) cells in vitro. They did, however, find that diesel exhaust particulate extracts were cytotoxic to the CHO cells and that extracts from cars from different manufacturers had different cytotoxicity. The cytotoxicity of diesel extracts was antagonized by serum, lung and liver cytosols and sulfhydryl agents in vitro. The detoxifying effects of the cytosols was enhanced by the addition of co-factors such as NADP which suggested an enzymatic detoxification in addition to protein binding. The authors suggested that similar detoxification of the toxic chemicals associated with diesel exhaust particulates may occur in vivo.

Li (380) found that the cytotoxicity of DCM extracts of exhaust particulate generated by a light-duty diesel engine was decreased in the presence of serum, bovine serum albumin, lung and liver cytosols and sulfhydryl compounds such as glutathione and cysteine. The results suggested that the cytotoxic activity of diesel exhaust particle extracts was reduced in vitro by reaction with serum and sulfhydryl compounds. Although the extent of in vivo inactivation of the cytotoxic or mutagenic activity of diesel extracts has yet to be shown, these data suggest that protein binding, chemical reactions and enzymatic metabolism may decrease their toxic effects.

Pereira et al (321) studied the effect of a methylene chloride extract of diesel particulate from small engine emissions on the induction of sister chromatid exchanges (SCE) in the bone marrow cells of mice. Three-month-old male mice were injected intraperitoneally with diesel particulate matter at 300 mg/kg body weight, or diesel extract at 800 mg/kg body weight. After a single IP injection, animals were sacrificed at 1, 2, 5 and 14 days. The bone marrow cells were isolated and the metaphases evaluated for the number of SCE and the mitotic index was estimated as the total number of dividing cells per thousand cells. Diesel particulate, when administered IP, resulted in a substantial enhancement in the number of SCE per metaphase and per chromosome in the bone marrow cells. In mice treated with diesel particulate matter and sacrificed 1, 2, 5 and 14 days post-treatment, the average frequency of SCE per metaphase in the bone marrow cells was 5.4, 11.3, 6.7 and 5.1, respectively. The average number of SCE in the bone marrow cells of solvent-treated control animals was approximately 6 per metaphase. Thus, it is evident that the genotoxic effect of diesel particulate matter appeared on the second day of treatment. By the fifth day post-treatment, the substances were no longer capable of inducing SCE. The findings suggest that diesel particulates and a methylene chloride extract contained genotoxic substances.

The drosophila sex-linked recessive lethal bioassay was used by Schuller and Neimar (322) as a screen for genetic hazards of diesel emissions. This bioassay is a useful system allowing for detection of most types of genetic damage and has been used to study the mutagenic effects of many gases and aerosols.

Two hundred 2 - 5-day-old, wild-type, male fruit flys were exposed to an airstream consisting of diesel engine exhaust gases diluted five-fold with filtered ambient air. Of the two groups studied, the number of recessive lethals found in both treatment groups was smaller than in the control groups, thus indicating that there was a lack of mutagenic activity exhibited by diesel exhaust. The particulate concentration was approximately 7.3 mg/m^3.

There was some particulate loss and this may have been the result of adsorption along the network of fine wire mesh in the cage or on the walls of the glass chamber.

Two factors should be considered in interpreting this data. First, the number of flies used in this test is capable of detecting moderate to strong mutagens exhibiting a mutation rate 3 - 5 times the background or control mutation rate. A weaker mutagenic activity of two times the background would not have been detected. Therefore, in order to detect any special mutagenic activity, a higher concentration of diesel exhaust would have to be administered for a more thorough assessment of the mutagenic potential found in diesel exhaust.

In order to establish a baseline to define the sensitivity of test systems in which there was either an absence or presence of an exogenous metabolic activant, two model compounds were employed by Curren et al (323). N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) is a compound which is genotoxic for most microorganisms and does not require metabolic activation. Benzo(a)pyrene (BaP), was also assayed to determine the sensitivity of the system in detecting compounds which require metabolic activation for expression of mutagenic and carcinogenic activity. The transformation and mutation frequencies induced by 10 mcg/ml of BaP were lower than expected and may have reflected either the poor solubility of the compound or the different response of the 3T3C cells with increasing BaP concentration. Such a decrease in the transformation frequency induced by high doses of polycyclic aromatic hydrocarbons has been observed by others.

Dichloromethane (DCM) extracts of diesel engine exhaust particulate and other environmental emissions such as from coke ovens, were tested at 7 dose levels with and without a source of exogenous metabolic activation. The DCM extracts of diesel particulate were all toxic at a minimum of one dose, while the three comparative samples showed no toxicity during the treatment period for the simultaneous mutagenesis transformation assay. An exogenous metabolic activation system was added, but had no effect on the toxicity of the Caterpillar heavy-duty engine sample, although it did reduce the toxicity of samples from Oldsmobile and Nissan diesel engines.

Historical laboratory data with the BALB/C3T3 cells in this study showed a remarkably low spontaneous transformation and mutation frequency, thereby allowing the system to detect chemicals with fairly weak biological activity.

Preliminary studies with MNNG and BaP showed that 3T3C cells can respond in a dose-dependent fashion for both mutagenesis and transformation. However, none of the emission extracts yielded a dose-dependent increase in either mutation or transformation frequency. The absence of a dose-response makes quantitative comparison among the extracts difficult.

Mitchell et al (288) used the mitotic recombinogenic assay utilizing a yeast, as well as the assay for forward mutagenesis using thymidine kinase in mouse lymphoma cells and sister chromatid exchange (SCE) utilizing Chinese hamster ovary cells. Testing was performed with and without the addition of a metabolic activator.

These results of testing in the yeast assay were neither reproducible nor dose-related. It was concluded that none of the diesel or environmental samples induced a recombinogenic effect on the yeast. It is interesting that none of the 7 polycyclic aromatic hydrocarbons requiring activation, including benzo(a)pyrene, were active in this assay.

4.5.4 Toxicology of Diesel Particulate

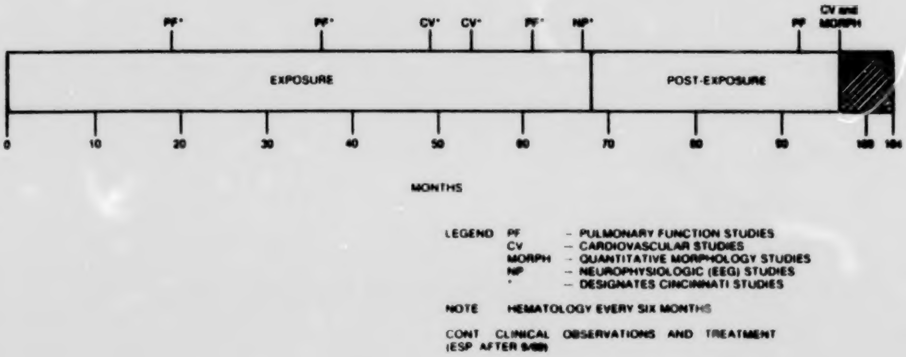
This sub-section presents findings related to the effects of whole diesel exhaust or its components to whole animals as a means of evaluating toxic effects as expressed by changes in tissue enzyme activity, morphology and/or histopathology as well as organ function, including carcinogenicity.

The EPA recently published the results of their long-term study of the effects of air pollutants in Beagle dogs (477).

Although this long-term study, in which female beagle dogs were exposed 16 hours daily for 68 months to various environmental pollutants including raw gasoline engine exhaust, is not specifically related to the diesel engine there were certain treatment groups, particularly those receiving SO_2 and sulphuric acid and NO_X, etc., which are of interest. Only those portions which are considered relevant to diesel exhaust toxicity will be discussed.

Of considerable significance was the continuing evaluation of the animals after the end of the 68 months of exposure during which time they were kept in "clean air" conditions. Figure 119 outlines the schedule of biological testing reported for this key study which was conducted from 1965 to 1974. Table 120 provides information on the exposure levels of the various groups included in the study. Table 121 lists the exposure concentrations for the various pollutants to which the animals were exposed over the 68-month study period. The concentrations of pollutants used were considered to be approximately 2 to 6 times greater than the average daily maxima occurring in several large U.S. cities during the same exposure period. Table 122 is a summary of the pollutant concentrations in the exposure chamber during the study.

No significant differences were seen in the concentration of collagen in lung parenchyma between exposed and unexposed animals, but significant differences in prolyl hydroxylase activity were seen between control lungs and lungs from animals which were exposed to a


PF*
PF*
CV*
CV*
PF*
NP*
PF
CV and MORPH
EXPOSURE
POST-EXPOSURE
0
10
20
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50
60
70
80
90
100
104
MONTHS
LEGEND PF – PULMONARY FUNCTION STUDIES
CV – CARDIOVASCULAR STUDIES
MORPH – QUANTITATIVE MORPHOLOGY STUDIES
NP – NEUROPHYSIOLOGIC (EEG) STUDIES
* – DESIGNATES CINCINNATI STUDIES
NOTE HEMATOLOGY EVERY SIX MONTHS
CONT CLINICAL OBSERVATIONS AND TREATMENT
(ESP AFTER

FIGURE 119

Schedule of biological testing from 1965 to 1974

(Source: Reference 477)

TABLE 120

Composition of Exposure Atmospheres and Abbreviations Used

Abbreviation	Atmosphere
CA	Control air
R	Non-irradiated auto exhaust
I	Irradiated auto exhaust
SO_X	0.5 ppm SO_2 + 100 ug/m^3 H_2SO_4 in control air
R + SO_X	Non-irradiated auto exhaust + 0.5 ppm SO_2 + 100 ug/m^3 H_2SO_4
I + SO_X	Irradiated auto exhaust + 0.5 ppm SO_2 + 100 ug/m^3 H_2SO_4
NO_L + NO_{2H}	0.2 ppm nitric oxide + 0.5 - 1.0 ppm nitrogen dioxide
NO_H + NO_{2L}	1.5 - 2.0 ppm nitric oxide + 0.2 ppm nitrogen dioxide

(Source: Reference 477)

TABLE 121

Estimated Exposure Concentrations (0800 to 2400 hr) daily for 68 months)[a]

Component	R	I	SO_X	NO_L+NO_{2H}	NO_H+NO_{2L}
CO (ppm)	100	100	--	--	--
HC as CH_4 (ppm)	24-30	24-30	--	--	--
NO_2 (ppm)	0.1	0.5-1.0	--	0.5-1.0	0.2
NO (ppm)	1.5-2.0	0.1	--	0.2	1.5-2.0
O_X as O_3 (ppm)	--	0.2-0.4	--	--	--
SO_X (ppm)	--	--	0.5	--	--
H_2SO_4 (ug/m^3)	--	--	100	--	--

[a] The actual measured concentrations are given in Table 122

(Source: Reference 477)

TABLE 122

Pollutant Concentrations in the Exposure Chamber Atmospheres During the Chronic Exposure Study (ppm (mg/m^3), mean ± sd)

Group	Atmosphere	Carbon monoxide CO	Hydrocarbons (as CH_4)	Nitrogen dioxide NO_2	Nitric oxide NO	Oxidants as O_3	Sulfur dioxide SO_2	Sulfuric acid H_2SO_4
1	Control air (CA)	4.9	2.7	0.04	0.04	0.02	0.03	....
2	Non-irradiated auto exhaust (R)	97.5 ± 10.0 (112.1 ± 11.5)	27.5 ± 4.4 (18.0 ± 2.9)	0.05 ± 0.02 (0.09 ± 0.04)	1.46 ± 0.42 (1.78 ± 0.52)	....	....	....
3	Irradiated auto exhaust (I)	94.5 ± 19.6 (108.6 ± 22.5)	23.9 ± 6.1 (15.6 ± 4.0)	0.94 ± 0.36 (1.77 ± 0.68)	0.19 ± 0.29 (0.23 ± 0.36)	0.20 ± 0.09 (0.39 ± 0.18)	....	....
4	$SO_2 + H_2SO_4(SO_x)$	....	....	....	....	....	0.42 ± 0.22 (1.10 ± 0.57)	0.02 ± 0.01 (0.09 ± 0.04)
5	Non-irradiated A.E. with $SO_2 + H_2SO_4(R + SO_x)$	98.4 ± 13.8 (113.1 ± 15.9)	27.4 ± 4.3 (17.9 ± 2.8)	0.05 ± 0.03 (0.09 ± 0.06)	1.51 ± 0.44 (1.86 ± 0.54)	....	0.48 ± 0.23 (1.27 ± 0.61)	0.02 ± 0.01 (0.09 ± 0.04)
6	Irradiated A.E. with $SO_2 \pm H_2SO_4(I + SO_x)$	94.6 ± 19.8 (109.0 ± 22.8)	23.9 ± 6.0 (15.6 ± 3.9)	0.89 ± 0.36 (1.68 ± 0.68)	0.19 ± 0.29 (0.23 ± 0.36)	0.20 ± 0.08 (0.39 ± 0.16)	0.42 ± 0.21 (1.10 ± 0.56)	0.03 ± 0.01 (0.11 ± 0.04)
7	Nitrogen oxides (high NO_2), ($NO_L + NO_{2H}$)	....	....	0.64 ± 0.12 (1.21 ± 0.22)	0.25 ± 0.06 (0.31 ± 0.08)	....	....	....
8	Nitrogen oxides (high NO), ($NO_H + NO_{2L}$)	....	....	0.15 ± 0.33 (0.27 ± 0.62)	1.67 ± 0.21 (2.05 ± 0.26)	....	....	....
	Nominal	100	24-30	0.5-1.0	1.5-2.0	0.2-0.4	0.50	(0.100)

(Source: Reference 477)

high concentration of NO and a low concentration of NO_2. Because there were no early measurements taken, it was impossible to be certain whether changes in collagen synthesis occurred early. It was suggested that may have been the case, but that by the time the animals had completed the exposure and subsequent maintenance in clean air that any initial alterations in connective tissue levels would have been obliterated. Prolyl hydroxylase is an enzyme involved in collagen synthesis and changes in its activity usually denotes changes in collagen synthesis. It was suggested that the tissues with the higher levels of prolyl hydroxylase were actively turning over more collagen and were in a dynamic state of repair and regeneration.

Pulmonary function testing was performed by the EPA Cincinnati Group (477) prior to initiation of the exposures and after 18, 36 and 61 months of exposure. Cardiovascular studies were completed after 48 and 54 months of exposure. Various pulmonary function tests were carried out at the different evaluation times and variably included single breath diffusion capacity, pulmonary compliance, total expiratory resistance, maximum breathing capacity, peak expiratory flow rate, pulmonary membrane diffusion and pulmonary capillary blood volume. Cardiovascular status was evaluated using ECG's, vectocardiograms, post-exercise ECG's, selected blood pressure and phonocardiogram determinations.

At 18 and 36 months following exposure to all experimental conditions, no statistically significant treatment effects were seen for any pulmonary function test performed, using an analysis of variance statistical approach. After 61 months of exposure, an ANOVA test of significance showed significant differences in the functional status among the different treatment groups, but only involved dogs which received exhaust as part of their treatment regimen. No changes were seen in animals who received SO_2 or NO_X's only. Additional statistical analysis indicated significant changes in some of the groups. For example, there was a decrease in peak expiratory flow rate in dogs receiving low NO plus high NO_2 treatment compared to the clean air group. Although there were changes in individual animals in cardiac electrophysiology, no consistent changes were observed for all animals in any one treatment group. The experimental atmospheres used did not appear to produce readily detectable dramatic cardiopulmonary responses following the 5-year exposure. Unfortunately, the methodology to measure changes in small airway obstruction which would be indicative of the development of chronic obstructive lung disease (COLD) were not available until near the termination of the study. The investigators concluded that two important features of response to air pollutants were demonstrated in the study. One was that exposure to air pollutants resulted in a gradation of individual responses even within the same treatment. The incidence of individuals responding was the most sensitive means of detecting treatment effects. Secondly, the exposed individuals, at the pollutant concentrations used, had a decreased ability to perform vital functions but not an absolute failure of subsystem components resulting in death or significant morbidity. They summarized their findings by suggesting that treatments associated with alveolar, parenchymal response included auto exhaust, auto exhaust plus SO_X, NOL plus NO_2H, and NOH plus NO_2L. The study was also thought to demonstrate that exposure to air pollutants produced the following aspects of lung disease: pulmonary hyperinflation, non-uniform distribution of inspired air, reduced diffusing capacity and elevated respiratory resistance. The cardiovascular responses indicative of disease included: cardiac arrhythmias, ventricular access deviation, bradycardia and premature ventricular contractions.

Gillespie reported on the cardiovascular and pulmonary function of the dogs after 68 months of exposure and two years after completing the exposure. Although there are too many details to be presented in this document a summary of the findings is given. As to the cardiovascular system, the authors suggested that the values obtained during and after exposure did not show any clear cut effects of air pollutants. The cardiovascular function of beagles from all groups studied 36 months after termination of exposure were similar and values were closely related to those of healthy beagles reported in the literature. There appeared to be no evidence of progressive cardiovascular disease which could unequivocally be associated with air pollutants.

It was also concluded that the pulmonary function studies supported the hypotheses that (1) exposure to low levels of specific air pollutants produced pulmonary injury and loss of pulmonary function and (2) the functional loss continued following termination of the exposure. All groups exposed to air pollutants demonstrated differences between their two year post-exposure function values and those obtained immediately after exposure and many of those of the control group. In general, automobile exhaust appeared to injure airways and parenchyma whereas SO_X, NO and NO_2 caused injury to the parenchyma. The functional abnormalities correlated in most instances with structural changes in the exposed groups. Those animals exposed to automobile exhaust, with or without added SO_X, showed substantial atypical epithelial hyperplasia in their small airways. Although less extensive, these same lesions were seen in the other exposed groups. The animals exposed to higher concentrations of NO_2 and SO_X had the greatest airspace size and parenchymal damage as shown by several quantitative morphometric measures. Probably the most significant finding was the fact that pulmonary function immediately after the 68 month exposure demonstrated only modest pulmonary function changes. Two years after the termination, many of the pulmonary function values showed an even greater difference from those of controls, which were of a type consistent with an emphysemic response. The investigator suggested that the animals that were exposed to SO_X and NO_2 had the most substantial functional changes at the parenchymal level.

One reviewer commented that he was surprised at the findings that SO_X produced only parenchymal damage. In view of the fact that SO_2 exposure was a mixture of SO_2 and sulphuric acid at a mass median of 0.6 um. SO_2 is water soluble and deposits in the upper respiratory tract and the sulphuric acid particles would grow by a factor of 2 or 3 in diameter as they entered the lung. Thus, he would have expected them to plate out on the bronchi as well. Some of the differences may be a result of the greater scrubbing effect of the longer nares in the beagle dog than in humans, which could result in larger particles being removed, with the remaining smaller particles depositing in the parenchyma and the very small airways. Furthermore, the dog is a frequent mouth breather.

Light and electron microscopic examination of lung biopsy tissue was carried out. The findings were complex but the investigators reported that there were areas of distended alveoli that may have represented "compensatory emphysema". There were no appreciable differences among specimens from the control, irradiated exhaust, or NOL plus NO_2H animals. Possible abnormalities were limited to occasional evidence of exaggerated goblet cell activation in the bronchioles, multiple layering of bronchiolar epithelium and evidence of perivascular fibrosis in all three groups. The other experimental groups were not evaluated.

Additional histopathology was completed on animals at necropsy and organs such as lung, liver, heart, kidney, etc., were weighed and examined histopathologically. No unexpected non-pulmonary findings were seen in any of the dogs. In dogs who had been in clean air for 32 to 36 months following 68 months of exposure to pollutants, there was enlargement of airspaces in proximal acinar regions, with and without increases in the number and size of intra-alveolar pores, and hyperplasia of non-ciliated bronchiolar cells. Proximal enlargement of air spaces was most severe, both subjectively and morphometrically in those dogs exposed to NO_X, SO_X or SO_X with irradiated auto exhaust. In contrast, hyperplasia of non-ciliated bronchiolar cells was most severe in dogs exposed to raw auto exhaust alone or with oxides of sulphur. The airspace enlargement and hyperplasia of the bronchiolar epithelium correlated with the functional impairment summarized above. The observations indicate that enlargement of proximal acinar airspaces, with some loss of intra-alveolar septa can develop in the absence of alveolar fenestrations. Two major toxicologic implications came from the study; (1) that the production of permanent lung damage by much lower concentrations of pollutants than had been previously reported were seen, and (2) the apparent lack of additive or synergistic effects between the oxidant gases and sulphur oxides.

In summary, the histologic and electron microscopic observations revealed that in lungs from five of the seven treatment groups, there was appreciable enlargement of distal airspaces which was centered on respiratory bronchioles and alveolar ducts and was associated with apparent loss of intra-alveolar septa, which the investigators considered to be analagous to an incipient stage of human proximal acinar (centrilobular) emphysema. Although it was of a mild degree compared to the clinically important cases of human emphysema, it was sufficient to cause abnormalities in pulmonary function. The two groups with the greatest decrease in alveolar surface density were the high dose NO_2 and SO_X groups which had higher than normal dynamic compliance at lower frequencies than the control group. This was interpreted as evidence of decreased lung elasticity. The authors also noted that it was somewhat surprising to find that the low concentrations of NO_X caused these emphysematous type changes. Other studies in the literature have shown that, for NO_2 alone, concentrations to produce moderately severe emphysema in rats was reported to be 28.75 mg/m^3 with continuous exposure for the natural life span of the animal, whereas in the present study 1.21 mg/m^3 of NO_2 and 0.31 mg/m^3 of the less injurious NO for 68 months at 16 hours a day produced what they considered to be mild, but definite emphysema. The presence of the airspace enlargement in lungs of dogs exposed to SO_X was unexpected. In the EPA study, airspace enlargement was caused by 1/10th the concentration of H_2SO_4 (0.09 mg/m^3), together with an amount of SO_2 (1.10 mg/m^3) comparable to that used in guinea pigs and monkeys in which these changes were not observed.

A large portion of the data have come from the GM diesel research group who have contributed a significant quantity of data from an impressive research program completed within a modest time frame. In discussing the GM data, it should be remembered that the conditions of animal exposure were the same for the various areas explored. These conditions are repeated frequently so that the reader is clear as to the experimental conditions used.

It is worthwhile emphasizing that although the GM group apparently considered the concentration of particulate exposure to be greater than expected for ambient urban conditions, except for exposure at 6 mg/m^3 (6000 mcg/m^3), most were at levels of between

0.25 and 1.5 mg/m^3 (250 - 1500 mcg/m^3), which are similar to those estimated to be present in the underground mining scene. Thus, from a particulate concentration point of view, the GM exposures are relevant to the concerns of this review.

A general description of the experimental conditions used in the General Motors Research Chronic Inhalation Diesel Exhaust Studies can be found in Reference 382. A summary of the inhalation chamber environmental conditions is given in Table 123.

Gross (326, 395) tried to resolve the question of a possible effect of diesel exhaust inhalation on lung function by studying the effects of long term (612 days) exposure to diluted diesel exhaust on lung mechanics and volumes in anesthetized male rats, using a body plethysmograph. This allowed the transpulmonary pressure, or the pressure difference between the air way and plural size of the lungs to be measured.

Lung air flow, volume of air breathed and respiratory rate were monitored in the body plethysmograph during spontaneous breathing by recording the flow-volume curve.

Although a significantly larger value for the relative normalized functional residual capacity, expiratory reserve and residual volumes in diesel-exposed animals might have been indicative of chronic obstructive lung disease they were considered similar to changes seen in other studies in which an emphysema-like condition was induced in rats by intra-tracheal instillation of elastase. However, the interpretation of these changes was contradicted by the mid-expiratory flow (MEF_{40}), MEF_{20} and the forced expiratory volume (FEV_1) values. Gross concluded (326) that if chronic lung disease was occurring, these indices would be expected to decrease in the diesel-exposed animals compared to the controls. The fact that they increased in the diesel group, suggesting an improvement in airway calibre, was not consistent with that expected on studies reported in the literature for human pulmonary disease. The majority of the measured parameters did not differ significantly between control and diesel-exposed groups.

TABLE 120

Inhalation Chamber Environmental Parameters[a]

	Control	1500 ug m^{-3}	750 ug m^{-3}	250 ug m^{-3}
Chamber temperature (°C)	21.5 ± 0.7	21.8 ± 1.0	21.6 ± 0.7	21.6 ± 1.1
Relative humidity (%)	48 ± 9	51 ± 9	48 ± 7	44 ± 10
Dilution ratio (approx.)	--	40:1	80:1	240:1
Nitrogen oxides (mg m^{-3})	0.05	9.2 ± 1.6	5.0 ± 1.2	2.1 ± 0.6
Carbon monoxide (mg m^{-3})	2.2 ± 0.6	7.9 ± 2.1	5.3 ± 0.9	3.4 ± 0.8
Carbon monoxide (mg m^{-3}) (corrected for ambient)	0.0	5.7	3.1	1.2
Aerosol concentration (ug m^{-3})	7 ± 9	1533 ± 346	796 ± 228	258 ± 87[b]

[a] Mean ± standard deviation over all measurements between 12/878 and 12/19/80

[b] 261 ± 178 including one filter of 8042 ug m^{-3} drawn during high exposure on 12/18/79

(Source: Reference 382)

It was concluded that this non-invasive pulmonary function testing of rodents was well suited for determining potential effects of chronic pollutant inhalation. The experimental advantage of following the same animals' responses over an entire exposure period was demonstrated. The deposited diesel particles provided a far greater burden than would usually be found in people forming the general population exposed to diesel exhaust on United States roads. However, the tests are limited in scope in that they do not serve all the factors and indicators of the many possible alterations that could occur in the lung when exposed to diesel exhaust.

It should be emphasized that the point of these studies was to look for symptoms of chronic lung disease and such lung function changes could take much longer to develop than the experimental time frame used in these tests.

Barnhart, Chen and Puro (329) investigated the impact of inhalation of diesel engine particulate in short-term and chronic inhalation experiments with Sprague-Dawley and Fisher 340 rats and Hartley guinea pigs. These two animals were chosen because they have differing sensitivities to toxic substances. Exposures of 250, 1500 and 6000 mcg/m^3 in ambient air, which corresponds to dilutions of 1:240, 1:40 and 1:10 dilutions of raw exhaust, were used.

Scanning and transmission electron microscopy were used to study the effectiveness of the phagocytic defense mechanism of the pulmonary alveolar macrophages (PAM), both in situ and in lavage specimens.

The use of the scanning electron microscope (SEM) permits the study of the 3-dimensional organization of the lungs and, at low magnification, allows areas and volumes of the compartments to be quantitated. After 15 weeks of exposure to exhaust particulate there appeared to be no significant difference in the relative amounts of parenchymal tissue among the three groups of rats.

Ten micrographs, taken at random and photographically enlarged, allowed determination of volume fractions and calculation of the percent of four cell types, PAM (pulmonary alveolar macrophages), Pn-I (pneumocyte-I), Pn-II (pneumocyte-II) and endothelial cells. Two significant differences were noted with exposure to 6000 mcg particulate/m^3 which resulted in a four-fold increase in the fractional volume percent of PAM and a 1.75-fold increase in the Pn-II over control values.

The remarkable uptake of diesel particulate by PAM was clearly seen using transmission electron microscopy (TEM). The particulate appeared to stay enclosed within membrane-bound structures within the cells rather than free within the cytoplasm or on the basement membrane. Interstitial cells, which were regarded as macrophages, were seen to contain particulate. The interstitial cells were located near the bronchial alveolar junction and probably illustrates part of the translocation of the particulate to the drainage sites of lymph and blood. The PAM cells were filled with particulate and were found on the epithelium of the terminal bronchioles, which indicates that these macrophages were preparing to exit the lung by way of the major clearing mechanism, the mucociliary escalator.

The Pn-I (pneumocyte-I) cells were also capable of incorporating particulate following diesel emission inhalation and this epithelial cell was filled with distinctively sized particulate in vesicles. Thus the normal epithelial cell, where the exchange of oxygen and carbon dioxide takes place, was breeched, in that the barrier was no longer intact. This may have occurred by way of phagocytosis, but it is more likely that the particulate was pinocytosed, and vesicles within the cell fused to produce a larger vesicle.

Consideration was given to the concept that particulate might penetrate or enter at weakened cell junctions between adjacent epithelial cells, which normally have very tight junctions. However, SEM and other analyses failed to reveal the presence of separated junctions between the particulate-stressed epithelial cells.

In guinea pigs, particulate was taken up both by PAM and Pn-I cells and appeared as discrete particles with average diameters of 0.05 microns and were in membrane-enclosed vesicles within the cells.

Characteristics of the pulmonary alveolar macrophage were obtained by broncholavage of the guinea pigs. It was suspected that without massage of the lungs or any other forcing the pulmonary macrophages seen in the broncholavage would be largely old and debilitated cells that had finished their job. Nevertheless, when these PAM cells were stained with trypan blue dye, they were shown to be viable. Other researchers have demonstrated that PAM cytotoxicity is present in vitro on exposure of lavage cells to fly ash. It was found that smaller particles of less than 2 microns were the most toxic. However, examination of cells in the present study failed to demonstrate any significant cytotoxic effect.

Exposed pulmonary macrophages, when cultured, showed marked ability to adhere to and spread on glass and plastic surfaces during a two-hour period. This was considerably more pronounced than macrophages from control animals. The response of the exposed PAM cells were dose-related. Although more work is needed to investigate the details of these findings, it would seem that the PAM cells are responding to the stress of diesel particulate and were more "activated" than the control PAM cells.

A notable finding was the tremendous size variation among the cells in the lavage specimens and also in the adherence of cells, which was particularly prominent following the 1500 mcg/m^3 dose. The control PAM cells had an average diameter of 13 microns. In contrast, cells from animals exposed to 250 mcg/m^3 dose measured 17 microns with a range of 10 - 24 microns and those from animals receiving a particulate dose of 1500 mcg/m^3 had an average diameter of 25 microns, with a range of 10 - 44 microns. This suggests that the surface areas of these enlarged cells were greater, which resulted in a greater uptake of particulate. Thus, there was a two-fold increase in diameter and a 3-fold increase in surface area in the PAM cells exposed for 8 months to the 1500 mcg/m^3 of diesel particulate.

The surface activity of the exposed PAM cells resembled that of controls but it was often more extreme. Roughing on the cell surface was prominent in cells from the 250 mcg/m^3 dosed animals and blebs and blisters were notable in more cells from animals dosed at the 1500 mcg/m^3.

A small cell type, other than PAM, was identified and appeared to be a granulocyte (eosinophil), which is a leukocyte with granules approximately 0.5 microns in size. There were 25% eosinophils in the lavage fluid from 1500 mcg/m^3 dosed animals, 14% in cells from the 250 mcg/m^3 exposed guinea pigs, compared to 3% in control lavages. Blood smears prepared from cardiac punctures of the same animals did not demonstrate the same kind of eosinophilia. Neutrophils, which might be expected if the response to particulate was an acute inflammatory reaction, were rare in any of the guinea pig lung lavages. However, it is well known that the guinea pig often produces eosinophils in increasing numbers following a chronic exposure to other substances.

There was an unusual group of large cells which were classified as being in the mononuclear PAM category. These bizarre cells comprised 20% of the lavage cell population, exclusive of the other cells. They resembled the "reactive" monocytes which have been identified in blood during certain chronic infections. These cells were, however, not observed in blood smears from exposed guinea pigs that had reactive cells in their tracheobronchial lavage fluid.

Because of the very careful handling of the broncholavage procedure, with no massage and/or pressure of the lung, it was assumed that the PAM cells in the lavage fluids were largely those already on the mucociliary escalator, as well as the cells that were free in the alveolar spaces. It is probable that these would include older and more debilitated cells that were in the process of making their exit from the lung into the escalator or into the lymphatic system. However, it is notable that these cells retained their viability. It is interesting to speculate on the number of cells remaining within the lung, particularly those that are fixed in the interstitial tissues or in the wall of the lung alveoli. The study demonstrated the importance of the lavage cells to retain their viability and functional capabilities even though they were heavily burdened with particulate.

As a continuing part of their investigations into the ultrastructural, biochemical and functional effects of diesel exhaust on guinea pigs and rats exposed to diesel exhaust particulate, Chen et al (330) examined bronchi-alveolar lavages from the animals exposed in the GM Diesel Exhaust Program. Exposures were clean air, or diesel exhaust at 250, 750, 1500 and 6000 mcg of diesel exhaust particulate/m^3 inhaled for a few days or chronically up to 18 months. Viability, according to dye exclusion, was 90 -99% in all specimens. The absolute number of alveolar macrophages increased 1.4 - 2 times over control values by 6 and 12 months exposure to 750 and 1500 mcg of diesel exhaust in guinea pigs. In rats, after 2 months exposure to 6000 mcg of diesel exhaust/m^3, the macrophages were 5 times the control levels. Except for the 250 mcg group, macrophage size also increased with the calculated macrophage volume increasing up to 4 times that of the controls. The diesel exhaust particulates were phagocytozed without cytotoxic effects since phagosomal membranes remained intact and macrophage lactic dehydrogenase activity was not present in the lavage fluids, which would have occurred had the cells lysed. Macrophages from 12-month animals exposed to 750 and 1500 mcg/m^3 of diesel exhaust had reduced staining for acid phosphatase, while the cell-free lavage fluid showed a 2 - 5-fold increase in enzyme activity. Comparison of equivalently-dosed animals revealed similar magnitudes and change in macrophage number and granulocyte recruitment. After diesel exhaust exposure, granulocytes became a significant percent of the free cell population by one month at 6000 mcg/m^3, two months at 750 and 1500 mcg/m^3 and 12 months at 250 mcg/m^3 of exposure. In the rat, the lavages contained neutrophilic granulocytes, which contrasted with the eosinophilic granulocyte mobilization seen in the guinea pigs. Lymphocytes appeared in the lavages from both species after two months exposure. Both granulocyte emigration and elevation in proteins at the high exhaust exposures were considered features of a classic inflammatory response, but it was noted that the 250 mcg/m^3-exposed animals, even after 18 months, elicited few and generally insignificant changes compared to controls. It was concluded that the defense capabilities of both rodent species appeared adequate to cope with the chronic diesel exhaust challenges given to them.

Weller, Chen and Barnhart (315) reported on the effect of diesel engine exhaust exposure on pulmonary alveolar macrophages in rats and guinea pigs. Animals were exposed to diesel exhaust particulate levels of 250, 1500 and 6000 mcg/m^3 for from one day up to 12 months. Age matched controls were concurrently exposed to clean air. Cytochemical techniques were used to identify macrophages and to determine if the uptake of diesel exhaust particles resulted in changes in lysosomal enzymes. Cells were evaluated by light microscopy after being cytocentrifuged onto glass slides and reacted for non-specific esterases using a naphthy-butyrate substrate and for acid phosphatase, using naphthyl AS-BI phosphate as substrate. Esterase staining confirmed the identity of the macrophages, with phosphatase staining permitting quantitation of enzyme activity. The pulmonary alveolar macrophages from control animals usually had a 98% positive reaction, whereas the macrophages from diesel-exposed animals were not as reactive as controls and the percent negative cells increased. For example, at day 1 exposure to 250 mcg/m^3, 38% of cells were negative, whereas exposure to 1500 resulted in 90% of the cells being negative. After 12 months exposure to 1500 mcg/m^3, 70% of the macrophages were negative. It appeared that reduced acid phosphatase activity was both time and exhaust-dose related. Although localized acid phosphatase was discernible in some cells the presence of black diesel exhaust particulate complicated the identification and interpretation of the data.

Schreck and co-workers (332) described the results of their chronic inhalation studies in rats and guinea pigs which were exposed to diluted diesel exhaust from a 5.7 litre 4-cycle indirect-injection Oldsmobile engine. Animals were exposed for 20 hours per day, 5.5 days per week for 106 weeks to nominal particulate concentrations of 250, 750 and 1500 mcg/m^3. The gas concentrations measured at the highest exposure level were typically 6.6 ppm of CO, 6.9 ppm of NO and 0.5 ppm of NO_2. The aerosol delivered to the chambers was similar in size to diesel exhaust particulate released into the atmosphere. The calculated weekly dose of diesel particulate delivered by inhalation is given in Table 124.

TABLE 124

Calculated Weekly Dose of Diesel Particulate

Diesel Aerosol Mass Concentration (mcg m^{-3})	Calculated Weekly Deposited dose per Rat[a] (mcg)
250	100
750	300
1500	600

[a] Assuming a minute volume of 200 mL and a total deposition efficiency of 30% of the inhaled aerosol.

(Source: Reference 332)

It was found that chronically exposed animals matured normally but histopathological work-up of tissue samples remains to be completed.

Strom (333) reported the results of bronchopulmonary lavage in the GM rats exposed to diesel exhaust at particulate levels of 250, 750 or 1500 mcg/m^3 for 26 - 48 weeks. There was no difference in the number or kinds of cells (alveolar macrophages, polymorphonuclear leukocytes, lymphocytes) between control and 250 mcg/m^3 exposed animals after either 6 or 11 months exposure. However, after exposure to 750 or 1500 mcg/m^3, the number of alveolar macrophages increased by 25 - 33 and 100 - 150%, respectively. Polymorphonuclear leukocytes, which were not found in lavage fluid from control rat lungs, were obtained in high numbers from rats exposed to 750 or 1500 mcg/m^3 for 11 months, or 1500 mcg/m^3 for 6 months of diesel exhaust. When identical amounts of particulate was administered over twice the length of time, the number of alveolar macrophages was increased by 25% over control values compared to 150% after shorter exposure to a higher concentration. This demonstrated that the pulmonary cellular systems responded primarily to the rate of submicron particles entering the lungs, rather than to the total load of particulate delivered to the lung. The probable cause of the observed effect was considered to be the continuous accumulation of excessive amounts of both "indigestible", carbonaceous particles and nuclei. When the macrophage renewal rate in the lungs is too slow to cope with the influx of particles, the macrophages become overloaded.

Dziedzic (4, 334) explored the possibility that diesel exhaust could be immunotoxic. It was thought that if the immune function was impaired animals could have an increased susceptibility to acute or chronic infections. Cell viability and differential counts of lymphocyte populations were carried out in guinea pigs exposed to 1500 mcg/m^3 of diesel particulate for 4 or 8 weeks and compared to age-matched controls. The data showed that the inhalation of high concentrations of diesel particles had no significant effect on proportions of B and T cells in lymph nodes, blood or spleen. As well, there was no effect on the viability of cells from the exposed groups of guinea pigs who inhaled the exhaust for up to 8 weeks when compared to controls. The author cautioned that more extensive studies employing more sensitive markers of immuno response are required before the question of possible interactions with the immune system could be definitively answered.

Other members of this same GM research group (335) reported on the pathologic alterations in rat lung after exposure to 6,000 mcg/m^3 of diesel particulate for periods ranging from 6 hours to 9 weeks. The alveolar macrophages or scavenger cells of the pulmonary system offer the first line of defense against inhaled foreign particles and this was observed in these studies, as well as an increase in Type II pneumocytes and an increase in the inflammatory cells within the alveoli and septal walls. Histochemical differentiation of alveolar macrophages and Type II cells (those cells presumably involved with the synthesis of surfactants) were both increased, with the latter being a somewhat unexpected finding.

Barnhart and co-workers (337) presented the results of morphometric, ultrastructural analysis of alveolar lungs from the guinea pigs exposed at the GM Diesel Laboratories. Gross inspection of lungs made it apparent which animals had received diesel exhaust exposure with such lungs being dusty-grey in colour with punctate blacker regions. Under light microscopy, the most notable signs of exhaust exposure was the scattering of pigmented macrophages occasionally clustered at the ends of the terminal bronchioles. Pigmentation was observed in the lymphatics near the bronchiolar-alveolar junctions. The pigment deposits increased in relation to both duration and exposure dose. By six months at the 750 mcg dose, some fibrosis was noted in the regions of macrophage clusters and evidence of focal epithelial Type II cell proliferation was present. The general ultrastructural organization of the alveolar lung was not appreciably altered due to diesel exhaust exposure. At diesel exhaust exposures above 250 mcg, there was an increase in the cellular composition of the interstitium of the lungs when compared to their matched control animals. Fibroblasts, monocytes, eosinophils, plasma cells and macrophages were identified more often within the perivascular and peribronchiolar interstitium than in alveolar walls. Hypertrophy and proliferation of Epi II cells were also noted. Detailed electron micrographic examination of lung tissue indicated that exposures to 250 mcg/m^3 of diesel exhaust/m^3 caused little or no change in the morphometric qualities of lung tissue after 24 months exposure. Although diesel exhaust particulate (DEP) was observed within the alveolar macrophages and Epi I cells at the 250 mcg dose, it was more prevalent at higher exposures. It was considered significant that regardless of the carrier cell type, the particulate remained sequestered within membrane-enclosed vesicles. This showed that the phagocytozed exhaust particulate did not escape into the cytoplasmic milieu. Furthermore, the exhaust particulate could not have been cytotoxic to the alveolar macrophages or the observed increase in the absolute numbers of those cells would not have occurred. The appearance of particulate within Epi I cells most probably resulted from particulate overload which the alveolar macrophages could not handle. Thus, the Epi I uptake of particles may represent a second line of defense against particulate matter.

Ultrastructural changes occurred as early as two weeks after exposure to the 750 mg dose, illustrating how dynamic and responsive the normal lung is to environmental stress. Thickened alveolar septa were noted for exposures over 250 mcg/m^3. The increased thickness was considered to be due to an increase in the number of interstitial cells, some of which were macrophages and eosinophils, and to an increased numerical density and hypertrophy of the Epi II cells. Although the arithmetic and harmonic mean tissue thicknesses of the air-blood barrier were occasionally increased, the morphometric diffusion capacity was not adversely affected. The investigators concluded that 250 mcg of diesel exhaust/m^3 through 24 months exposure elicited insignificant tissue changes, except for alveolar macrophage uptake of diesel exhaust. As well, the lung responded rapidly to diesel exhaust challenges greater than 250 mcg/m^3 by increasing the cellularity of all types of alveolar lung cells. Partial adaptation occurred in normal guinea pigs during chronic exposure to diesel exhaust illustrating the normal lung's potential for repair and defense.

This same group (338) examined the structural physiology of airways near gas-exchanging alveoli using scanning electron microscopy. Diesel exhaust particulate was identified as individual particles (Figure 120) as well as being adherent to epithelium and as irregular patches of particles at airway bifurcations (Figure 121). Proximal airway, characterized by an epithelium of secretory and ciliated cells, had even larger agglomerates of particulate, particularly in the higher dose animals. These agglomerates tended to be larger than would be expected for diesel exhaust particulate, and may have been an admixture of granules and proteins. Terminal bronchioles were noted to be crowded with macrophages and granulocytes exiting the lung. In the heavily-exposed animals, more pneumocyte II cell clusters were present at the bronchiolar-alveolar junctions than were seen in control animals. The alveoli opening off terminal bronchioles contained more particulate matter than did other alveoli. Although not absolutely identified, the data suggested that these resulted from the diesel exhaust particulate burden.

The mutagenic and carcinogenic effect of many polyaromatic hydrocarbons were frequently reduced not through the parent molecule, but through the biological metabolites, usually arene oxides, being transformed by the action of aryl hydrocarbon hydroxylate (AHH) and epoxide hydrase (EH). Chen and Vostal (339) examined the

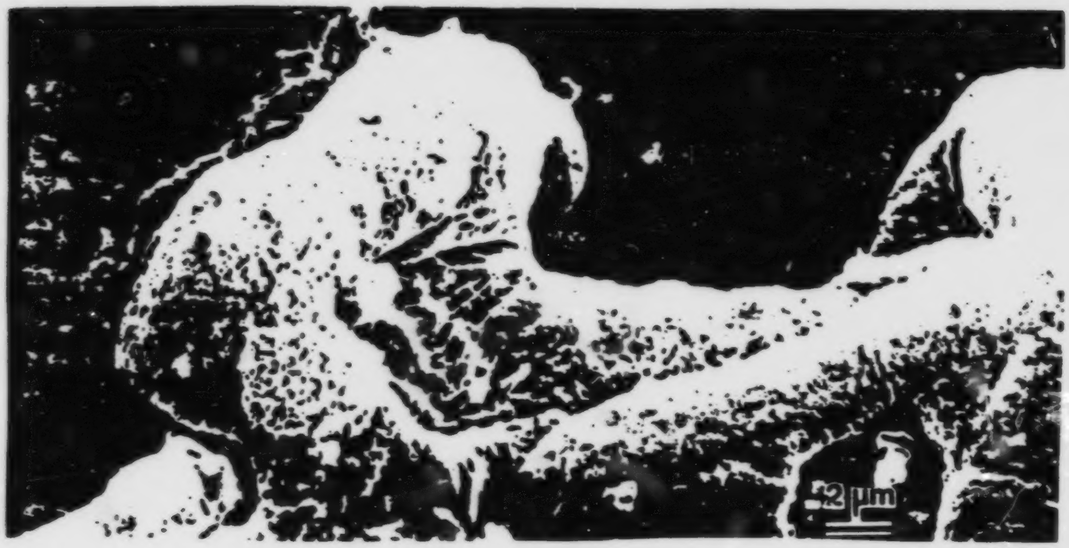

FIGURE 120

Terminal airway of guinea pig exposed to 1500 mcg DE for 12 months. Note patches of particles whose individual size is 0.1 um and could be DE deposits.

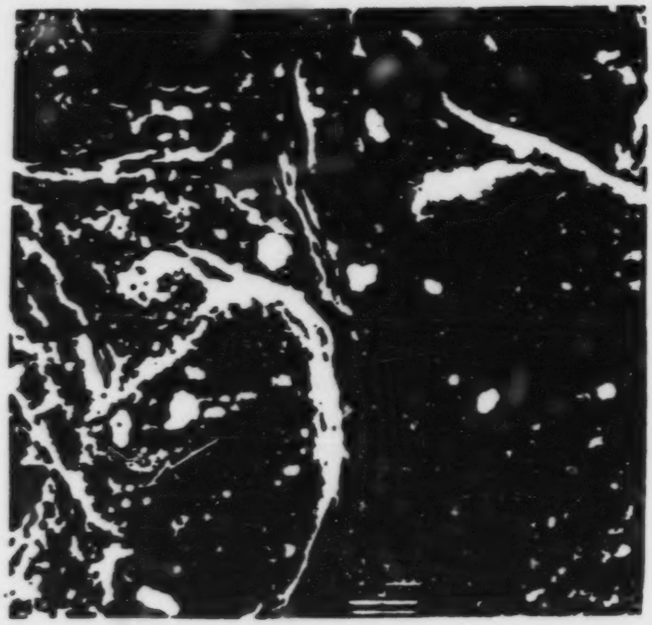

FIGURE 121

Terminal bronchiole adjacent to alveolus in rat exposed to 6000 mcg DE for 2 months. Small dust particles are 0.1 um in diameter and probably DEP

(Source: Reference 338)

effect of inhalation exposure to diesel exhaust on the induction of AHH enzyme in lung and liver tissues. Male rats were exposed to 750 or 1500 mcg/m^3 of diesel particulate from a 5.7 litre GM diesel engine 20 hours per day, 5.5 days per week, for up to 9 months, or received repeated intraperitoneal or direct intratracheal instillation of diesel particulate extracts. No effects of long-term inhalation exposure were observed on liver microsomal AHH activity. A slight decrease in lung microsomal AHH activity was recorded in rats after 6 months of exposure at the high dose. The intraperitoneal injection of an equivalent dose of extractable hydrocarbons also caused no increase in either lung or liver microsomal AHH activity. These results demonstrated that the hydrocarbons adsorbed on inhaled diesel particulate were not effective as enzyme inducers, possibly because of their lack of availability for distribution in the body. A direct intratracheal administration of the diesel particle extract required doses as high as 6 mg/kg body weight before activity of the induced enzyme in the lung could be barely doubled. The induction was slow and occurred selectively in the lung only, again suggesting that diesel particulate extract probably does not adsorb easily into the lung circulation and is not distributed to other organs.

In a recent publication, the GM diesel research group (487) reported the results of exposing rats, which were pre-loaded with diesel particulate matter to ^{14}C-tagged (^{14}C)-n-hexadecane diesel particulate for 40 - 45 minutes, followed by examination of lung tissues for ^{14}C content of diesel particulate immediately after nose-only exposure to isotopic particulate and at intervals up to 12 months following exposure. The animals were pre-exposed to either 0, 250 or 6,000 mcg/m^3 of diesel particulate generated by a single-cylinder diesel engine for 20 hours per day for 7 to 112 days. Table 125 provides a summary of the exposure conditions as well as the lung content of diesel particulate matter at the end of the chronic exposure period.

A control group with no previous exposure to diesel exhaust was included. During the brief radioactive exposure, the diesel particulate level was 6.4 ± 0.3 mg/m^3. Figure 122 shows the particulate

TABLE 125

Estimated Cumulated Diesel Particulate Content in Lungs of Fischer Rats Based on the Data of Vostal et al (1982)

Group	Diesel Particulate Concentration (ug/m^3)	Exposure Duration (d)	Lung Content of Diesel Particles (mg)
Controls	-0-	-0-	-0-
2	250	52	0.2
3	250	112	0.5
4	6000	7	0.7
5	6000	62	6.5
6	6000	112	11.8

(Source: Reference 487)

retention, which was measured for the pre-exposed animals who had initial lung contents of diesel particulate ranging from 0.2 to 11.8 mg.

Lung clearance calculations suggested that the lung clearance time for the control group was 76 ± 5 days compared to 100, 123 and 153 days for groups 2, 3 and 4, respectively. Additional calculations suggested that in groups 5 and 6, who were exposed to 6,000 mcg/m^3 of particulate for 62 or 112 days, there was a non-clearing compartment of particulate which represented 45 and 88% of the lung burden, respectively. Figure 123 provides a plot from which the residual retention in the lung can be estimated based on the particulate lung burden of diesel particulate. It was concluded that the retention of inhaled particles could be interpreted as impaired clearance due to either prolonged exposure to high concentrations of diesel exhaust or accumulation of particles in sequestered macrophage aggregates within the lungs.

Pepelko (324) reported on work carried out during 1979 - 1980 as two main groupings, the first being non-oncogenic toxicology experiments and the second, those emphasizing carcinogenesis and mutagenesis. Cats were exposed to diesel exhaust from a Nissen 6-cylinder engine at 6.4 mg/m^3 of particulate for one year and

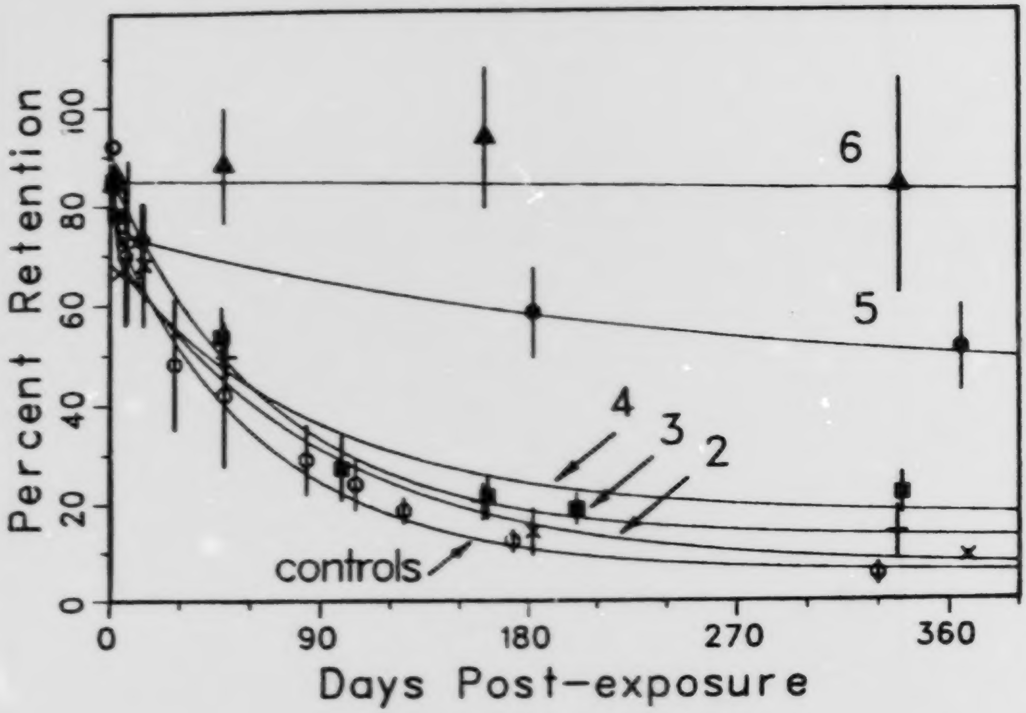

FIGURE 122

Retention of ^{14}C diesel exhaust particles inhaled for 45 minutes in Fischer rats with a previous diesel exposure history. The lung content of diesel particles in each group of test animals is summarized in Table 125. The line curves are computer-fitted by non-linear regression. Each data point represents the mean value of four measurements and the vertical bars are standard deviations.

(Source: Reference 487)

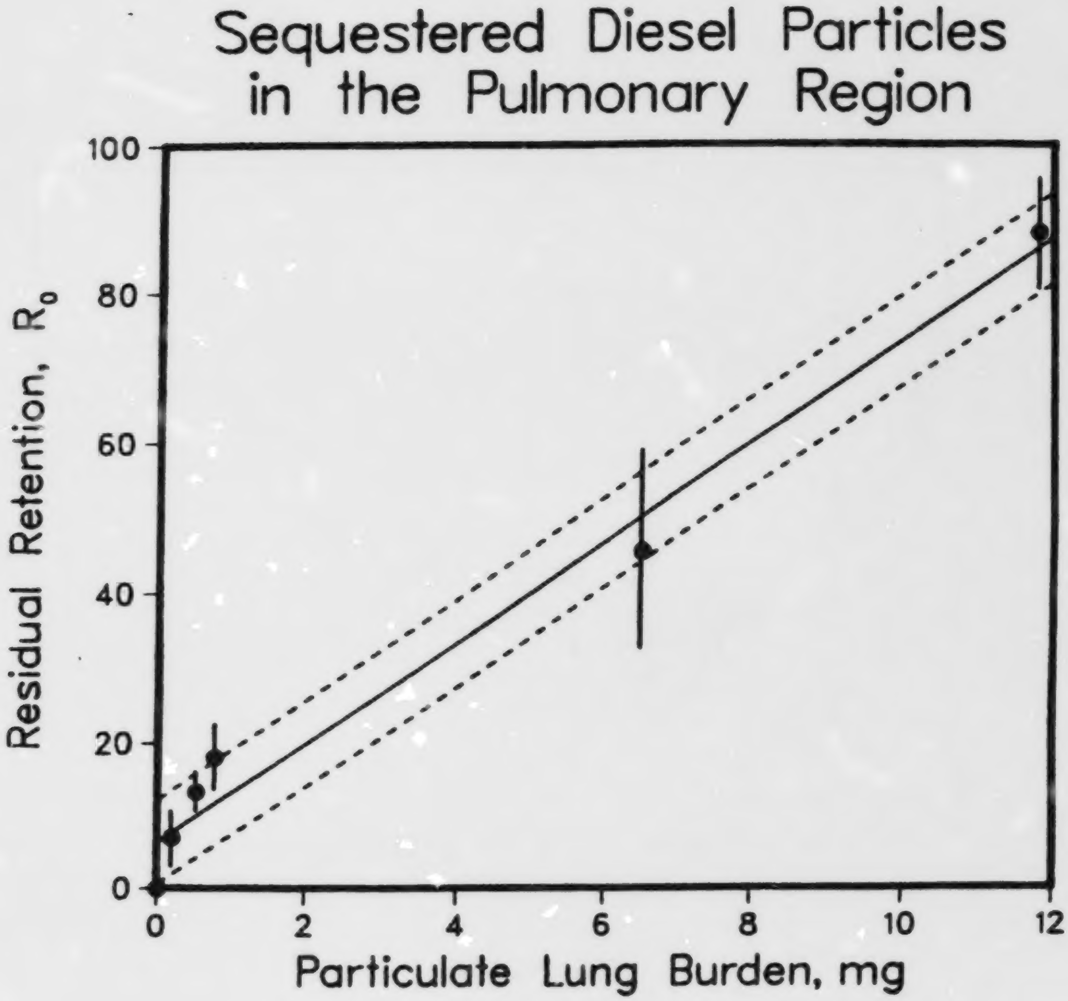

FIGURE 123

The residual fractions of sequestered particles. R_0, obtained through the curve-fitting procedure, were plotted against the initial diesel particulate lung burden. Asymptomatic standard errors are shown as vertical lines. The dashed lines are 95% confidence bands for the regression line.

(Source: Reference 487)

pulmonary function tests completed. During the second year of exposure, 19 of the original 21 male cats were exposed to 11.7 mg/m^3 of particulate, 4.37 ppm NO_2, 5.03 ppm SO_2 and 33.3 ppm CO with total hydrocarbons of 7.72 ppm (325). Twenty male cats served as controls. Pulmonary function testing was carried out on anesthetized cats in a complex pulmonary testing laboratory which included a variable pressure plethysmograph. After the first year of exposure, no significant differences were found in mechanical properties, diffusing capacity, uniformity of distribution or ventilatory performance. However, after two years exposure, changes were measured. Statistically significant reductions in the inspiratory capacity, vital capacity and total lung capacity were measured, with normal values being found for ventilatory function, which indicated that a lesion was present which restricted breathing, but did not cause airway obstruction and loss of elasticity. The restrictive disease measured by these pulmonary function tests is compatible with a diagnosis of pulmonary fibrosis of the interstitital or intra-alveolar type. As well, chronic inflammation, interstitital edema or vascular engorgement may have been present. The presence of an impaired diffusing capacity was considered to support the diagnosis of interstitial lung disease.

Pathological observations which have been reported by other investigators (references 11 and 12 from Reference 325) included accumulation of black pigment in the macrophages, in the interstitium, hyperplasia of the alveolar lining cells, with focal thickening of the interstitium, and interstitial pneumonitis. Pathological examination of the cat's lung was not completed at the time this report was made, but the pathological descriptions by other investigators would be consistent with the physiological finding of restrictive lung disease reported by Pepelko and co-workers (325).

The same group also exposed mice for 3 months and Chinese hamsters for 6 months (324). Exposure to diesel exhaust during the critical period of gestation did not result in a detectable increase in birth defects in either rats or rabbits.

There was an increase in lung tumour incidence in female mice only. These findings were questionable, however, because of a lower than normal tumour incidence in the controls.

Golden Syrian hamsters were also used. When rats were used, the liver island test was utilized as a relatively short-term (3 - 6 months) test for liver cancer and the induction of gamma glutamyl transferase enzyme was used as a marker of cancer-altered phenotype.

Several enzyme studies were done on the strain A mice exposed 6 months to diesel exhaust containing 6 mg/m^3 of particulate. The cytochrome tests for P-450 and the AHH induction test were done, but no important changes were detected.

Inhalation studies were completed using components of exhaust and in this study rats were exposed up to 6 months to either 2.5 ppm NO_2, or 6 mg/m^3 of carbon particulate, or a combination of both. Rats were exposed up to 28 days to as much as 4 ppm of acrolein followed by measurement of pulmonary function and pathology. Acrolein was selected as one of the particulate components of the exhaust because it is known to be the most irritating of the organic compounds present in diesel exhaust. Data from these studies is either still being gathered or is being analyzed.

Pepelko et al (327) evaluated the effects of exposure to automotive diesel exhaust containing 6.4 mg/m^3 particulate and its effect on the pulmonary function of the Chinese hamster. Details of other pollutant levels were not given.

Adult male chinese hamsters were exposed to diesel exhaust at a dilution of 1:18 for 8 hours a day, 7 days a week for 6 months. The animals were examined for vital capacity, alveolar volume, carbon monoxide transfer factor and volume pressure curves. Pathological examination of the lung tissue has not yet been completed. Elevated lung volumes at 0 and 5 cm of water for the volume pressure curve showed that the volume, expressed as a percent of vital capacity, indicated a possible emphysema-like condition in the diesel-exposed animals. This is consistent with the significantly reduced carbon monoxide transfer factor found in these same animals.

Orthoffer et al (328) used a Strain A mouse lung tumour model to examine the carcinogenicity of diesel exhaust. This mouse tumour model has a known percentage of lung tumours that it generally develops in the course of its short lifetime, and it was considered a good animal to use to contrast against similar animals exposed to diesel exhaust and the use of intraperitoneal injections of urethane.

A 6-cylinder Nissan automotive diesel engine with a Chrysler transmission mounted on an absorption dynamometer was used in the study. The engine was cycled continuously for 20 (Study 1) or 8 hours daily (Study 2). The diesel engine exhaust mass was 6.39 mg/m^3 during the 8 hours/day, 7 days/week study. Animals in the 20 hour/day group had exposure to irradiated exhaust from the diesel engine, with 25 of the same Strain A mice being placed in a non-irradiated exhaust exposure chamber, 25 in a control air chamber and 25 were positive controls which were injected intraperitoneally with urethane (20 mg/mouse) and placed in a control air chamber. The mice were exposed for 7 weeks to diesel exhaust and then held in control air chambers for 26 weeks until sacrificed. A-type mice were divided into 6 groups with Group 1 being given 235 ug, Group 2 117 ug, and Group 3 47 ug of diesel particulate by intraperitoneal injection. Group 5 was a positive urethane control, Group 4 was a clean air control and Group 6 received no injections.

In Study 1, no gross changes in condition or appearance were observed in the mice. Exposure to diesel exhaust did not affect the growth rate compared to the controls. The animals exposed for 30 weeks were uniformly black in appearance. Their lungs were black, sometimes grossly. It appeared that the black areas outlined the intermediate and small airways of the lungs. Examination under an ordinary microscope revealed that macrophages containing large amounts of black granular material had accumulated around the terminal bronchioles.

The mice who received the i.p. injections of particulate, as well as the diesel exhaust exposed mice were sacrificed 30 weeks after the first treatment. There was a 31% incidence of lung tumours with an average number of 0.28 tumours per mouse in the untreated controls and a 37% incidence of tumours with an average number of 0.42 per mouse in mice injected with 0.85 sodium chloride solution. In the urethane-injected animals many had multiple tumour nodules. In some cases the nodules had coalesced into a large tumour mass making it impossible to determine the number of initial nodules.

The mice that had been injected with diesel particulate intraperitoneally 3 times weekly was estimated to be the same as the inhalation dose, assuming a 50% retention rate. The highest dose group received 705 ug per week. There was no evidence of toxicity in any of the treatment groups as measured by general appearance and growth rate. The number of tumours in mice injected with diesel particulate and the control mice did not differ. The male mice that were given 30 and 38 week exposures, designated A-1 to A-3, showed no dose-related effects, or gross or microscopic lesions which could be attributed to diesel exposure.

Generally, the results of these studies were negative with the exception of the urethane-injected animals. These positive control mice were males, whereas the promotion animals were all females. Literature references report a lower spontaneous tumour formation in females which would tend to make the test more significant.

In these experiments, no increase in the incidence of lung tumours was found following as much as 9 months of inhalation exposure to diesel engine exhaust. However, the addition of urethane in low doses did elicit a positive response to the combination of urethane and diesel exhaust. The role of urethane as an initiator and/or promotor for lung cancer is known. Neither the urethane nor the diesel exhaust exposures alone produced a marked increase in lung nodules. Exposures to the two together, however, did produce a modest increase in pulmonary adenomas in the Strain A mouse. Diesel particulate was present in the lungs and draining lymph nodes 26 - 30 weeks after the exposure.

The residence time of the polycyclic hydrocarbons in the lungs after cessation of exposure because its elution rate from diesel particle under physiological conditions has not been determined. The presence of particulate material in the lymph nodes indicated that the diesel particulate was transported from the alveoli to the interstitium and the lymphatics. Previous research found migration of free carbon particles through the type 1 cells to the interstitium where they were rapidly phagocytozed by the interstitial macrophages. Also, the presence of carbon in the hyalar lymph nodes, 12 hours after appreciable amounts of carbon had been taken up by interstitial macrophages, indicated that the carbon particle reached the lymph nodes in the free state.

The methods used gave a higher concentration of particulate than would have been found under more severe and controlled environmental conditions. It was clear that the clearance mechanisms of the lung were unable to remove part of the diesel particulate as indicated by the presence of particulate in the lungs 20 - 30 weeks after the cessation of exposure.

Earlier work had demonstrated the development of pulmonary adenomas in Syrian Golden Hamsters after intratracheal instillations of automobile exhaust. The higher tumour rate was thought in part to be due to the failure of the mucociliary defense mechanism to clear the airways. However, the instillation procedure did not correspond to the human situation of inhalation and the instilled material was administered as a condensate. Inhaled substances are usually gaseous or bound to carrier particles likely to be removed by mucociliary action.

The number of mice with tumours were compared and the results are shown in Table 126. Overall, there was no indication that exposure of Strain A mice to diesel engine emissions resulted in an increase in lung tumour incidence. On the contrary, the studies consistently showed that tumour rates were decreased in exhaust-exposed animals.

Kaplan et al (331) presented a summary of a sub-chronic study of the effects of diluted diesel exhaust exposure on rats, hamsters and mice, at a particulate level of 1500 mcg/m^3 for 20 hours per day, 7 days per week, for up to 4½ months. At various times body weights, organ weights, and organ histopathology were examined. A portion of the animals studied were sacrificed after 6 months of recovery. It was

TABLE 126

Effects of Inhaled Diesel Engine Emissions Upon Lung Tumor Incidence in Strain 'A' Mice

Treatment	Age at Sacrifice (Months)	Illumination During Exposure	Sex	# of Mice with Tumors/ # of Surviving Mice	P Values
Clean Air	9	Light	M	10/44	
			F	11/43	
			M+F	21/87	
Diesel Exhaust	9	Light	M	5/37	NS
			F	4/43	<0.05
			M+F	9/80	<0.05
Clean Air	9	Light	M	32/38	
+			F	34/37	
5 mg Urethane			M+F	66/75	
Diesel Exhaust	9	Light	M	26/39	<0.10
+			F	16/36	<0.001
5 mg Urethane			M+F	42/75	<0.001
Clean Air	12	Light	M	22/38	
Diesel Exhaust	12	Light	M	11/44	<0.01
Clean Air	9	Dark	M	28/97	
			F	31/140	
			M+F	59/237	
Diesel Exhaust	9	Dark	M	13/111	<0.01
			F	9/139	<0.001
			M+F	22/250	<0.001

(Source: Reference 323)

found that exposure to diesel exhaust did not affect animal health as measured by mortality, growth weight or organ weights in any of the three species studied. Strain A/J mice which were exposed to diesel exhaust showed no increase in the average number of adenomas nor in the incidence of pulmonary adenomas compared to controls. After 3 months of exposure, particulate was distributed diffusely and in foci in the alveolar spaces of the lung, both in free form and in macrophages and in the regional lymph nodes. After six months of recovery, partial removal of particulate from the lungs and redistribution into focal accumulations had occurred. Most of the particulate was found inside macrophages or within protean matrices. Clearance of the particulate by lymphatic channels was evident in both exposed and recovered animals. In exposed and recovered animals of each species, alveolar fibrosis associated with large accumulations of particulate was minimal and not considered of clinical significance. Lesions in organs other than the respiratory system were minimal and of similar incidence in control and exposed animals.

In a later report, White and Garg (336) found that the Type II cells released considerably increased quantities of surfactant which accounted for the morphologic change in the phagocytic alveolar macrophage taking on a more foamy appearance. These responses were seen not only at the very high diesel dose of 6,000 mcg/m^3 for 9 weeks, but also when animals were exposed to 1500 mcg/m^3 for 2 years. After 9 weeks exposure to the high dose of diesel e[illegible]he accumulation of macrophages and Type II cells resulted in the formation of alveolar complexes that seemed to be preferentially located near the terminal bronchioles. The lesion was considered to be similar to that of macrophage alveolitis which can be produced in animals repeatedly exposed to coal dust.

Along a similar vein, Peirano (340) measured the effect of diesel exhaust administered by inhalation to mice 8 hours a day, 7 days a week for 6 or 8 months (at a particulate level of 6 mg/m^3) on the activity of xenobiotic metabolizing enzymes. Lung and liver microsomal AHH and liver microsomal cytochrome $P_{448/450}$ levels were determined. No statistical differences in the liver microsomal cytochrome $P_{448/450}$ levels or AHH activities were seen between animals exposed to clean air or diesel exhaust. Similarly, no significant increases were seen between the two treatment groups in the lung microsomal AHH activities. These data further suggested that the bioavailability of the diesel exhaust organics, when adsorbed onto diesel carbon particulate, are not readily available for general distribution in the body. To circumvent this problem, animals were given an intraperitoneal injection of a diesel exhaust extract at a maximum tolerated dose. As well, groups of animals received the known enzyme-inducing compounds phenobarbital or 3-

methylcholanthrene. Both phenobarbital and 3-methylcholanthrene resulted in the induction of the respective enzyme systems in both sexes, but the injection of the diesel exhaust extract caused a significant increase in the liver cytochrome $P_{448/450}$ levels in male mice only. As well, the increase in activity was smaller than that seen for the two other inducers.

On the other hand, Lee, Suzuki, Lee and Dixon (341) determined the specific activities of aryl hydrocarbon hydroxylase (AHH) and epoxide hydrase (EH) enzymes in rat lung, liver and male reproductive organs after inhalation exposure to diesel exhaust. The diesel exhaust was generated by a Nissan engine and fed into an animal exposure chamber after diluting 13 to 1 with air. The diesel emission contained 14.2 ppm of total hydrocarbon and adult male rats were exposed to the diluted exhaust for 20 hours per day for 42 days. Although liver exhibited the greatest overall AHH activity among the organs tested, the percentage increase over control values was highest in the prostate gland. AHH induction in liver and lung occurred on day 33 of exposure and the maximum AHH induction was observed on day 42 with a 1.4 and 1.3-fold increase in lung and liver, respectively. EH activity was not changed significantly in any of the tissues tested.

Mauderly and his group (342) completed chronic rat exposure studies in which groups of rats and mice were exposed by inhalation to diluted whole diesel exhaust for up to 307 hours during a 12-week period. Although the diesel engine used was from a small car, the animals were exposed to concentrations of approximately 4300, 1050 and 250 mcg/m^3 of diesel particulate matter. After 1, 3, 6, 9 and 12 weeks of exposure, animals were withdrawn and evaluated for biochemical and cytological indices, tracheomucociliary clearance velocity, defenses against bacterial infection, immunological responses, respiratory function and morphology. Health effects from the exposure were considered to be minimal, although a mild inflammatory reaction occurred early, but was resolved by 12 weeks. Particle clearance in the trachea and deep lung was slowed. Table 127 gives a summary of mean exposure concentrations of the various components of the diesel exhaust. Although there were various

TABLE 127

Summary of Mean Exposure Concentrations and Cumulative Particle Exposures for Animals Exposed for Different Times

	Weeks of Exposure Before Withdrawal				
	1	3	6	9	12
Total Exposure Time, hours	32.5	102.5	172.0	205.0	307.5
High Level Group					
Particle Conc., ug/m^3	4675 ± 360[a]	4560 ± 670	4810 ± 690	4610 ± 800	4260 ± 1110
Cumulative Particle Exposure, mg hr/m^3	151	466	822	943	1310
CO Conc., ppm	8.8 ± 0.6	918 ± 1.1	11.8 ± 2.3	11.7 ± 1.9	11.5 ± 2.6
CO_2 Conc., ppm	1810 ± 540	1710 ± 120	1770 ± 100	1870 ± 200	2080 ± 400
Hydrocarbons Cone, ppm C	6.3 ± 3.7	4.7 ± 1.4	4.2 ± 1.1	3.9 ± 1.0	4.0 ± 0.8
SO_2 Conc., ppm	0.016 ± 0.004	0.028 ± 0.016	0.024 ± 0.012		
NO Conc., ppm	1.18 ± 0.16	1.13 ± 0.07	0.96 ± 0.23	0.80 ± 0.29	0.80 ± 0.25
NO_2 Conc., ppm	0.05 ± 0.01	0.06 ± 0.01	0.4 ± 0.5		0.4 ± 0.4
NH_3 Conc., ppm	2.3 ± 0.5	2.9 ± 0.5	2.7 ± 0.5	2.6 ± 0.7	2.5 ± 0.7
O_3 Conc., ppb	13.6 ± 4.6	16.1 ± 3.5	15.2 ± 2.2	13.8 ± 3.4	14.6 ± 3.1
Medium Level Group					
Particle Conc., ug/m^3	950 ± 150	1040 ± 250	1180 ± 290	1110 ± 310	1030 ± 340
Cumulative Particle Exposure, mg hr/m^3	30.6	107	203	229	317
CO Conc., ppm	2.3 ± 0.6	3.0 ± 0.7	3.9 ± 1.1	3.8 ± 0.9	3.7 ± 1.1
NH_3 Conc., ppm	3.5 ± 0.6	3.8 ± 0.3	3.3 ± 0.6	3.0 ± 0.8	2.9 ± 0.9
Low Level Group					
Particle Conc., ug/m^3	240 ± 30	250 ± 60	270 ± 60	250 ± 70	230 ± 73
Cumulative Particle Exposure, mg hr/m^3	7.75	25.7	46.1	51.7	70.8
CO Conc., ppm	0.8 ± 0.4	1.2 ± 0.3	1.6 ± 0.6	1.6 ± 0.6	1.5 ± 0.6
NH_3 Conc., ppm	3.2 ± 0.3	3.9 ± 0.6	3.4 ± 0.8	3.2 ± 0.8	3.2 ± 0.8
Chamber Control Group					
Background Particle Conc., ug/m^3	--	--	22 ± 15	33 ± 20	39 ± 20
CO Conc., ppm	0.5 ± 0.4	0.8 ± 0.4	1.0 ± 0.5	1.2 ± 0.7	1.1 ± 0.6
NH_3 Conc., ppm	2.8 ± 0.3	3.1 ± 0.4	2.7 ± 0.6	2.8 ± 0.5	2.8 ± 0.7

[a] All values are given as mean ± standard deviation of average of daily observations

[b] Hydrocarbon cutter overloaded in SO_2 analyzer

[c] NH_3 conversion to NO occurred in NO_2 converter. NO_2 values greater than NO.

[d] No determination.

(Source: Reference 342)

changes, such as an increase in the LDH of lavage fluid, in the high-level group and glutathione reductase activities were elevated in a dose-dependent manner after 3 weeks of exposure, there was an adaptive process which appeared to occur in the lungs of the exposed rats. For example, early indications of a mild inflammatory response seen in airway washings after 3 weeks, were gone by 12 weeks, although increased numbers of macrophages were measured. Generally, no significant changes in rat or mouse liver cytochrome P-450 levels were seen in any exposure group at any sacrifice time. Exposure of mice to diesel exhaust appeared to reduce both liver and intestinal aryl hydrocarbon hydroxylases after 3 and 6 weeks of exposure. However, at 12 weeks the levels in exposed animals were not significantly different from control values. There was no indication of any irreversible lung damage resulting from any level of exposure to diesel exhaust, as measured by the biochemical indices used in this study.

Heinrich et al (343, 344) reported early findings of a small-scale inhalation study using Golden Syrian hamsters exposed to diesel exhaust emissions. Eleven biochemistry and 7 hematological indices were measured repeatedly throughout the study as an indicator of the biological effects of diesel exposure.

As well as the Syrian hamsters, two groups of 20 rats were exposed for 18 to 24 months to the same diesel exhaust and were subjected to lung function testing as well as determination of the impact of diesel exhaust inhalation on the clearance of radioactively labelled iron oxide aerosol.

In order to demonstrate a carcinogenic effect, a higher level of carcinogens in the atmosphere was used to compensate for the short life-span of the hamsters in comparison to the long lifespan of the human.

Exhaust was generated by a Daimler-Benz DB 200 D Diesel engine mounted on an electrical brake dynamometer using diesel fuel containing 0.36% sulphur. Table 128 gives the average values of the exhaust components during the study period. The concentrations of 14 particulate PAH's was measured and found to be low, i.e. 7 mcg of BaP

TABLE 128

Analytic Average Data on Emission Concentrations During Exposure

	Measurement			
	In The Chambers		Before The Chambers	
	A	B	A	B
CO (ppm)	16.9 (± 3.0)	17.9 (± 3.3)	16.3 (± 3.2)	18.0 (± 3.4)
CO_2 (vol%)	0.63 (± 0.14)	0.67 (± 0.14)	0.52 (± 0.14)	0.57 (± 0.16)
SO_2 (ppm)	4.7 (± 1.6)	4.9 (± 0.9)	7.9 (± 3.8)	7.0 (± 1.4)
C_NH_M (ppm)	8.5 (± 3.4)	8.9 (± 2.8)	5.3 (± 3.8)	4.2 (± 1.7)
CH_4 (ppm)	2.1 (± 0.8)	2.5 (± 0.9)	1.0 (± 0.33)	1.0 (± 0.32)
C_NH_M-CH_4 (ppm)	6.3 (± 3.0)	6.3 (± 2.6)	4.3 (± 3.7)	3.3 (± 1.4)
NO (ppm)	15.8 (± 7.1)	15.6 (± 6.9)	17.6 (± 8.5)	16.0 (± 8.8)
NO_X (ppm)	16.3 (± 7.2)	15.9 (± 6.5)	17.7 (± 10.2)	16.4 (± 10.3)
NO_2 (ppm)	0.45 (± 0.42)	0.40 (± 0.28)	0.10 (± 0.09)	0.21 (± 0.27)
O_2 (vol%)	20.0 (± 0.7)	19.5 (± 0.6)	20.0 (± 0.7)	19.1 (± 0.5)
Particles (mg/m^3)	--	4.2 (± 0.3)	--	--

A: Exhaust Without Particles

B: Total Exhaust

(Source: Reference 344)

per g of particles. The mass median aerodynamic diameter of the particulate matter going into the chambers was 0.1 um.

The Syrian hamsters were exposed for 7 - 8 hours per day, 5 days a week for two years in inhalation chambers. Three exhaust dilutions were used in the 5 month inhalation study at ratios of 1:3, 1:5 and 1:10. One-half of each group received the total exhaust and the other half were exposed to the gaseous exhaust only, with no particulate present. One group of animals inhaled clean air only.

Because it was likely that the incidence of tumour production in the diesel exposed animals would be low, an attempt was made to produce a base-line tumour induction rate by applying a known carcinogen at the beginning of the inhalation exposure permitting investigation of the influence of the exhaust inhalation on this baseline rate. Each of 3 exposure groups of 288 hamsters were subdivided into 6 subgroups of 48 each, and pretreated in the following manner:

1) no additional treatment (controls)
2) 2 mg pyrene in 20 weekly intratracheal instillations of 0.1 mg each (second controls)
3) 2 mg dibenzo(a,h)anthracene, intratracheally instilled as under (2)
4) 6 mg dibenzo(a,h)anthracene, intratracheally instilled as under (2), but 0.3 mg each
5) 1.5 mg diethyl nitrosamine per kg body weight, subcutaneously injected
6) 4.5 mg diethyl nitrosamine per kg body weight, subcutaneously injected

The red blood cell count was somewhat decreased in all exposure groups compared to controls. However, the difference was significant only for those groups receiving the gaseous phase of the exhaust alone, without particulate. There was some variation in hematocrit and packed cell volume, but on the whole, these findings continued throughout. Methemoglobin and carboxyhemoglobin values of the exposed animals appeared to be higher than those of the controls, particularly in the highest dose groups. All of these values still lie in a range, however, where there is no serious risk to the transport of oxygen.

When the white blood cells were examined, the count was slightly lower in all exposure groups, in some cases even significantly, but no dose-dependent effects were observed.

Extensive enzyme measurements were carried out, including SGOT, SGPT and alkaline phosphatase. There was no clear indication of organic lesions, in the kidneys, liver or heart. However, an increase in the BUN level pointed to a kidney insufficiency when the animals were exposed to total exhaust. However, on histological examination of the organs, including heart, kidney, liver, spleen, there were no abnormal findings. There was no difference in the weights of these organs compared to the weights from control animals.

In the five-month experiment, the wet and dry weight of lungs of sacrificed test animals were examined. In all three dose groups the lung wet weight, expressed as a percentage of the body weight, of animals exposed to total exhaust, was significantly increased in the two highest dose groups. This was also the case for those animals which received intratracheal instillations of the two chemicals. However, it should be noted that those animals which only inhaled the gaseous phase of the exhaust and received no particulate had lung wet weights almost identical to controls. No difference could be detected between the animals with and without instillation of pyrene and dibenz(a,h)anthracene. Histopathological examination indicated that the main cause of the increase of lung weight resulted from the inflammed, granulomatous and proliferative alterations of the lungs of the exposed animals.

Life-time examinations are on-going in animals exposed to a particulate concentration of 4.2 mg/m^3. In each group, 25 animals were examined at 5 months and SGOT, SGPT, LDH and alkaline phosphatase measured. All were significantly increased in the animals exposed to total exhaust. BUN was markedly higher with the total protein content of the serum being higher, although when examined by electrophoresis, each fraction did not differ significantly from controls. These assessments will be repeated every 4 -5 months until the animals have died.

The detailed data from the 24 month exposures was not available, but the summary information available pointed out two particularly conspicuous findings. One was that the respiratory tract tumcur rate produced by the subcutaneous injection of diethylnitrosamine (DEN) was enhanced in hamsters exposed to exhaust atmospheres (Figure 124). The second finding was that the lung clearance of radioactive aerosols in the chronically treated rats was reduced (Figure 125).

Because of the concern referred to earlier, for the increased health effects of diesel use in coal mines, Karagianes, Palmer and Busch (345) completed a chronic toxicology study using 20 months of concomitant exposure to coal dust and diesel exhaust aerosols by inhalation in rats. The animals were 18 weeks old at the beginning of exposure and five groups were used as indicated in Table 129. The diesel engine used was a 3-cylinder, 43 bhp motor which was run on an automatic cycling system with the engine idling at 1400 rpm for 20

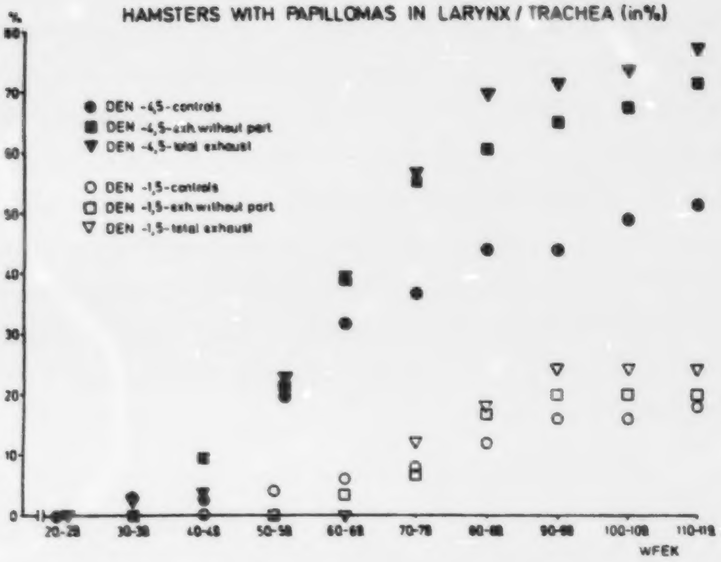

FIGURE 124

Papillomas in Pre-Treated Syrian Golden Hamsters

(Source: Reference 344)

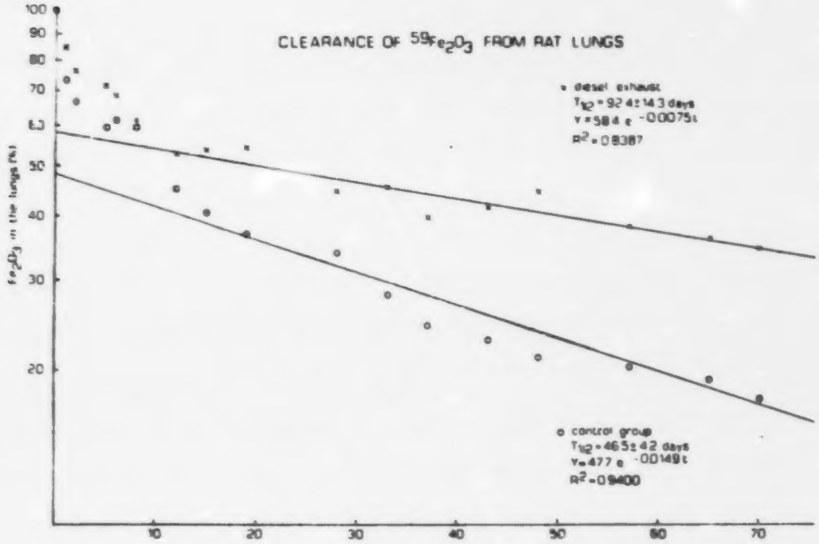

FIGURE 125

Lung Clearance of $^{59}Fe_2O_3$ Aerosols in Rats

(Source: Reference 344)

TABLE 129

Experimental Protocol for Exposing Rats to Coal Dust and/or Diesel Exhaust

Group	Exposure Regimen[A]	Concentration (mg/m^3)[B]
1	Diesel-engine exhaust	8.3 ± 2.0 soot[C]
2	Diesel-engine exhaust plus	8.3 ± 2.0 soot[C]
	Low-concentration coal dust	5.8 ± 3.5
	Total Particulate	13.5 ± 4.0
3	Low-concentration coal dust	6.6 ± 1.9
4	High-concentration coal dust	14.9 ± 6.2
5	Controls	Room Air

(A) 6 hr/day, 5 days/week; 40 rats/group

(B) Mean ± standard deviation over 20 months

(C) Soot concentrations are result of controlled CO level (50 ± 3 ppm)

(Source: Reference 345)

seconds, accelerated to 1800 rpm with no load for 20 seconds, decelerated to 1400 rpm with a load of about 9 kw for 80 seconds, accelerated to 1800 rpm as the load was removed and then decelerated to 1400 rpm to begin the cycle again. Air was added to the exhaust so that a dilution factor of approximately 35:1 was achieved, with a CO level of 50 ppm. The fuel injector tips of the engine were filed to increase the CO content without affecting NO_2 so that a simulated worn or poorly tuned engine situation was obtained which resulted in a CO/NO_2 ratio of 10:1 in the exposure chambers. Therefore, the animals were exposed to the TLV level of 50 ppm for CO continuously with an NO_2 content of about 4 -6 ppm. SO_2 and aliphatic aldehyde levels were below the detectable level of 1 ppm. The diesel fuel was industrial 2-D grade. Micronized bituminous coal was added to 3 of the chambers and had an MMAD of 2.1 micrometers and a GSD of 2.7. The diesel exhaust soot had an MMAD of 0.71 um and a GSD of 2.3.

The percent respirability was estimated to be 50% for the coal dust and 95% for the diesel exhaust particulate. Six rats per group were killed after 4, 8, 16 and 20 months of exposure and gross necropsy and histopathological examination was completed for these animals and any that died during the study interval (345).

No significant effect on body weight or on mortality patterns was observed for any of the coal or diesel-engine exhaust exposed groups compared to control rats. As well, there were no significant changes in hematocrit, RBC or differential leukocyte counts. There was a 4 -5% increase in carboxyhemoglobin (COHb) saturation in animals exposed to diesel exhaust as compared to control rats.

Gross examination of animal lungs showed the control rats to be normal whereas significant lesions were observed from all exposure groups. Lungs of coal dust exposed rats had clear evidence of coal dust accumulation and those exposed to the high coal dust concentrations had more pronounced lesions than those in the low dose group. In general, the degree of severity in both groups was related to the length of exposure.

The appearance of the lungs from rats exposed to either diesel exhaust alone or diesel exhaust plus coal dust were similar and were blacker in colour than the animals who received coal dust alone. The degree of discolouration increased with the length of exposure.

Histopathological examination of the rat lungs indicated lesions which were indicative of simple CWB (Coalworkers Pneumoconiosis) which included accumulation of black aggregates of inhaled particles in alveolar macrophages, alveolar histiocytosis, interstitial fibrosis and alveolar emphysema. The particle accumulation occurred at the level of the terminal bronchioles and followed the airway to the respiratory bronchioles and alveolar ducts. The deposited soot and/or coal dust extended along the adjacent alveolar septa and within adjacent alveolar sacs. Longer-exposed rats showed larger macules and panacinar emphysema.

Statistical analysis of the data showed that after 20 months exposure there were no significant differences in the severity of the lesions between the diesel exhaust, diesel exhaust plus coal dust and the high concentration coal dust exposure groups. No malignant respiratory tumours were found in any of the groups in this study (345).

Laurie et al (346) reported on studies conducted in the EPA labs in Cincinnati. The objectives were to measure any behavioural consequences of diesel exhaust exposure in adult and neonate animals. Previous reports that the exposure of rats to 6 mg/m^3 diesel exhaust for 20 hours per day 7 days a week significantly reduced two types of activity, spontaneous locomotor activity in adults (SLA) and pivoting in neonates.

Rats were exposed for 8 hours per day from 6:30 AM to 2:30 PM, 7 days per week to diesel exhaust generated by a Nissan CN6-33 engine. The raw exhaust was diluted with filtered air in a ratio of approximately 1:20, which resulted in a particulate matter concentration of 5.97 ± 0.17 mg/m^3 during the 16-weeks of exposure.

The exposed adult rats had a lowered spontaneous locomotor activity. Rats exposed to diesel exhaust for 8 hours a day showed a typical increase, and then a subsequent decrease of SLA over time and were significantly less active than the controls at one or four weeks of the experiment.

Based on reports in the literature CO, sulphuric acid, nitrogen oxide, nitrogen dioxide and sulphur dioxide were ruled out as causative effects of the reduction of SLA in adult rats. The components of the exhaust that were responsible for the observed effects on the behaviour of adult rats was either the hydrocarbons, particulate matter or perhaps a combination of some of the single constituents found in diesel exhaust.

When the neonatal rats were exposed to diesel exhaust particulate, the results suggested that permanent sequelae may be associated with the life history of the neonates. It seems that diesel exhaust given during development led to considerably less activity during adult life when compared to control groups.

In this particular study, unusually low activity levels were displayed by both control and exposed rats. Unfortunately the exposure was during the hours when rats are most quiescent and least active. Nighttime is the more active time for these animals. Neither the controls nor the exposed groups showed any signs of illness or any other reason for the failure of normal activity, especially when running on the wheel. Therefore, the evidence in this particular experiment cannot be taken as positive or negative evidence of the health hazards of diesel exhaust exposure.

Animals exposed to diesel exhaust had a great deal of difficulty acquiring a single bar-pressing task. This was considered further evidence that exposure to diesel exhaust during development may produce long-lasting changes in animal behaviour, although the nature of this behavioural deficit is not clear. It is impossible to attribute the effects of the neonatal exposure to any particular constituent of the diesel exhaust and unfortunately there has been little research done by other laboratories to compare with this study.

Laurie and Boyse (346) showed a clear association between neonatal diesel exhaust exposure and decreased adult spontaneous locomotor activity. As well, their work indicated that when neonates were exposed to diesel exhaust, differences in the acquisition of relatively simple tasks such as pressing a bar when the animals were 15 months of age resulted.

This study was an advance on the earlier report (347) in which the animals were assessed as to the effects of diesel exhaust on the development of the nervous system by measuring the somatosensory-evoked potential (SEP) and visual-evoked potential (VEP).

When the control rats and diesel-exposed neonates were compared, there were significant differences detected for developing SEP and VEP. It is not clear what caused the differences, but the exhaust exposure may have effected both the central and peripheral nervous systems. There were slower latencies in the various peaks which may indicate decreased myelination of both the central and peripheral nervous systems.

There was concern that there would be a toxic effect from the diesel exhaust which would alter or retard development during the time of rapid development around day 35. However, toxic effects which alter or retard development, would produce a larger absolute difference between the control and exposed animals as far as the wave forms are concerned around day 35 than on the other days tested. Specifying the particular processes affected by diesel exhaust without further study under exposure conditions is difficult because each developmental stage is highly dependent on the successful completion of previous events. Disruption at any point in the development of these neonatal rats can result in permanent changes.

Electrophysiological data reported in this article suggested that there are long-term behavioural alterations produced in neonatal rats that have been exposed to diesel exhaust and these are related to the failure to develop a normal, functioning nervous system.

As part of their overall research program on the toxicity of diesel exhaust, this group under the same experimental conditions described above, exposed mice from weanling age through to sexual maturity to diesel exhaust, after which the animals were mated and the dams continued exposure through pregnancy, parturition and weaning of offspring (348). The offspring were then reassigned to experimental groups and continued under their respective exposure for 15 months, at which time survivors were sacrificed. Clean air control and diesel exhaust test mice were assigned to three groups: Group A, to test for tumour initiation activity, was administered butylated hydroxytoluene as a promoting agent weekly for 13 months; Group B, used to test for promoting or cocarcinogenic activity, were administered an early single dose of urethane; and Group C were used to test for whole-carcinogenic activity and were given neither initiator nor promotor and were exposed to either clean air or diesel exhaust atmospheres. There were no treatment-associated differences in the numbers of dams producing litters, or in the average litter size, therefore there was no apparent effect of diesel exhaust exposure on the parental mice from weaning to parturition or on reproductive function. Neither was there any significant effect on neonatal survival to weaning. Although no statistically significant differences were noted, the authors suggested that the data showed a trend toward a greater incidence of pulmonary adenoma and carcinoma in mice chronically exposed to diesel exhaust. However, as the overall pool group comparisons showed a 9.8 versus an 8.7% incidence, the significance is questionable. The incidence and/or degree of severity of several lesion types were consistently and dramatically greater in diesel-exhaust exposed mice of both sexes than in the control animals. The lesions included alveolar macrophages, black alveolar pigment material, cholesterol clefts, perivascular and peribronchiole mononuclear cells, focal fibrosis, alveolar interstitial thickening, rhinitis and black pigment in the mediastinal lymph nodes. The results suggest that diesel exhaust does not have significant activity as an initiator or promotor when given in combination with BHT or urethane or as a whole carcinogen.

Slaga and Treplett (349) attempted to determine the skin carcinogenic, co-carcinogenic, tumour-initiating and promoting activities of diesel emission particle extracts. Several dose-response studies were completed and it was shown that there was an excellent dose response relationship for the initiating ability of BaP at 14 weeks using Sencar mice. A similar dose-response relationship was found for the tumour-initiating activity of a Nissen diesel sample, but using milligram quantities for initiation. Caterpillar diesel and cigarette smoke condensate were, for all practical purposes, 0, whereas Mustang gasoline and Olds diesel emissions gave extremely low values, although they were above background. Roofing tar, Nissen diesel and coke oven samples all gave moderate activity at high doses (1 - 10 mg), but when compared to BaP were relatively low.

In a later report from the same laboratory, Clapp and co-workers (350) published survival and tumour induction incidences in Sencar mice surviving one year of treatment with a dichloromethane extract of diesel exhaust collected from the engines indicated in Table 130. TPA (12-0-tetradecanoylphorbol-13-acetate) and BaP (benzo(a)pyrene) were inclued in the study protocol. The numbers of surviving mice were significantly reduced by TPA alone and all experimental groups given TPA reflected this treatment with decreased survival compared with controls. Lung tumour incidences varied with treatment groups but were not different from controls and TPA alone. Tumours of other organs were observed randomly in the treated groups but no consistent increased incidence could be associated with any particular treatment. It should be noted that

TABLE 130

Survival and Tumor Inductions in Sencar Mice Surviving 1 Year Given Diesel and Gasoline Particulate Emission Extracts to the Skin

			Tumors					
			Skin					
Treatment	Sex[a]	No. surviving mice (%)	T-positive (paps) (%)	No. paps/ No. surviving	T-positive carcinomas	Lung (%)	Misc (%)	Leuk-emias (%)
Untreated controls	F	100	0	--	0	3	0	3
	M	97	0	--	0	0	0	0
TPA	F	67	19	0.23	0	4	0	0
	M	71	4	0.04	4	4	4	0
BP + TPA	F	8	74	3.10	0	6	13	0
	M	56	100	4.70	14	4	0	0
OLDS + TPA	F	74	27	0.38	3	14	3	0
	M	95	42	0.67	3	19	0	6
Nissan + TPA	F	47	94	4.50	11	0	5	0
	M	46	89	3.40	5	5	0	0
VW + TPA	F	68	54	1.00	8	0	4	0
	M	75	50	1.00	3	3	0	0
OLDS (no TPA)	F	97	0	--	0	0	3	0
	M	97	0	--	0	3	3	0

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[a] F = female; M = male

(Source: Reference 350)

application of the Oldsmobile diesel extract without TPA resulted in no difference in the number of survivals or tumour formation compared to untreated controls. The Oldsmobile extract application only was applied twice weekly for the duration of the study, at a dose of 4 mg/mouse per week. In the other study groups, the animals were given one 10 mg/mouse exposure to the diesel extract, followed by twice weekly applications of the initiator, TPA.

Nesnow and co-workers (351) have published a more detailed report of their skin carcinogenesis studies of diesel emission extracts using the Sencar mouse. In summary, it was found that benzo(a)pyrene, coke oven main and roofing tar samples were positive in both sexes as tumour initiators, tumour promotors and complete carcinogens. Four diesel samples were positive as tumour initiators including Nissan, Oldsmobile, VW Rabbit and Mercedes Benz.

DePass et al (352) found that DCM extracts of diesel particulate, when applied to the dorsal skin of male mice, have little, if any, tumour initiating or tumour promoting activity.

Campbell et al (353) exposed young adult mice to diluted diesel exhaust for varying periods of time from a single 2-hour exposure to 8 hours/day for 321 days.

Mice were challenged with respirable, airborne pathogens which included, beta-hemolytic Group C Streptococcus pyogenes isolated from an animal lesion, an infective mouse-adapted influenza virus and Salmonella typhimurium. Prior to exposure to the infectious organisms, mice were housed in chambers where they were exposed to diluted light-duty diesel engine exhaust or to purified air. After the infectious exposure, the mice were housed for observation in cages and received single-pass filtered, conditioned air.

The chamber atmosphere contained 6 - 7 mg/m^3 of total suspended particulate, with the average concentration over the first year being 6.4 mg/m^3. The average concentration of NO_2, which was irritating to the respiratory tract and caused impaired resistance to infection by bacteria, was approximately 2.8 ppm

Short, intermediate and prolonged exposure to diesel exhaust resulted in enhanced susceptibility, with impaired resistance, to the infective organisms which terminated with lethal effects of bacterial infection which were qualitatively consistent throughout the experiment.

Both radiated and non-irradiated diesel exhaust was used. Non-irradiated diesel exhaust was clearly toxic to the systems responsible for coping with the inhaled Streptococcal infection, and the sensitivity was greatly increased to infection, even after very brief exposures of 2 - 6 hours.

The effect observed with respect to bacterial infections was absent in the diesel or NO_2 experiments when the viral pathogen was used. This was clearly apparent only in one of the three experiments testing acrolein, which was very active with bacterial infectivity. The limited data analyzed to date infers that the difference is probably due to different mechanisms.

The two sub-acute Salmonella experiments were inconclusive. The mortality rate was too high in the controls and consequently a proper comparison with animals that died after exposure to Salmonella could not be made.

The tests involving acrolein and NO_2 clearly indicated that both agents caused a marked increased in susceptibility to bacterial infection, which was particularly true for Streptococcus. Nitrogen dioxide was somewhat less toxic than acrolein. With the influenza virus neither acrolein nor nitrogen dioxide seemed to affect the defenses of the animals.

Diesel exhaust particulate from an Oldsmobile engine was administered to hamsters by intratracheal instillation for 15 weeks by Shefner and co-workers (354). The individual animals were sacrificed when they appeared moribund and an autopsy was completed on each animal. Diagnoses were based on pathological examination of tissue sections.

The results of the dose-range experiment showed that:

(1) diesel particles and diesel particles plus ferric oxide at the doses tested did not increase mortality above control levels.

(2) high doses of both preparations decreased weight gain in the first few weeks of treatment, but had no appreciable effect on weight gain by the end of treatment.

(3) there was no significant non-respiratory tract pathology in the treated animals.

(4) lesions of the lung were common in treated animals and included the following findings:

(i) adenomatous hyperplasia was more severe and extensive in diesel particulate treated animals than in the vehicle control animals.

(ii) adenomas were more numerous and larger in the diesel particulate treated animals than in controls.

(iii) metaplasia to ciliated epithelium and squamous metaplasia occurred in some diesel particulate treated animals, but not in the vehicle controls.

(iv) severe, multi-focal reactive pneumonitis with hyperplasia, with evidence of atypia, occurred in diesel particulate treated animals, but did not occur in the vehicle

Brune (403) investigated the effect of the percutaneous application of automobile exhaust condensates in the development of squamous-cell papillomas and carcinomas in mice. The sebaceous gland suppression test, which has been shown to be predictive of the carcinogenic activity of fractions from cigarette smoke condensate, was used as an investigative tool. The authors confirmed the results of others that the carcinogenic activity of automobile exhaust condensates resides in the polycyclic aromatic hydrocarbon fractions, of which BaP accounted for about 9% of the activity. As well, the sebaceous gland suppression test was predictive of carcinogenicity and was positive for those condensate sub-fractions which contained carcinogenic PAH's.

The incomplete results of a respiratory carcinogenicity study of diesel emission particles and various extracts of other PAH-containing materials by the weekly intratracheal administration for 15 weeks to hamsters was reported by Shefner et al (355). After twelve months of weekly treatment, randomly selected animals were sacrified and subjected to post-mortem and histopathological examination. Group mean organ weights of test hamsters showed no significant effects as compared with their solvent controls. Various lesions of the lung were noted which appeared to be treatment-related and generally reflected the quantity of particulate material administered. In general, inflammatory reactions and granuloma were more prevalent in diesel particle, diesel extract and coke oven extract-treated animals than in cigarette smoke or roofing tar-treated groups. Adenomatous hyperplasia and papillary hyperplasia were either found at a low incidence or not observed in solvent and colony control hamsters and from a trace to a mild degree in solvent plus ferric oxide exposed animals. The incidence and severity of these lesions was greatest in both diesel particulate groups and lowest in the diesel extract group, as well as in the other treated groups. In general the frequency and severity of hyperplastic and metaplastic changes in the lung was greatest in the two diesel emission particle test groups. Whether any of the lesions will progress over the life-span of the surviving hamsters which continue on test, remains to be evaluated.

In similar studies using mice, Laurie et al (356) administered the different regimens indicated in Table 131 by intratracheal instillation three times weekly for eight weeks with animals being evaluated at 9 months of age. As can be seen in Table 131, positive controls receiving urethane were included. From the data presented in Table 131, it is clear that a significant increase in the number of lung adenomas per mouse and percent of mice with tumours occurred in the positive controls. There were no consistent, statistically significant differences between the exposed and control groups with respect to either number of adenomas per mouse or number of mice with adenomas for the extract-treated animals. It was concluded that either the carcinogens present in the test substances were very weak or that an insufficient concentration reached the lungs to produce a positive result.

Wiester, Iltis and Moore (387) investigated the effects of inhaled diesel exhaust on pulmonary function, growth rate, electrocardiogram and histology of various tissues in guinea pigs. Using a Nissan CN6-33 engine and #2 diesel fuel, they found the exhaust to contain pollutant concentrations as indicated in Table 132. Animals were exposed continuously for varying periods of time, 20 hours per day, 7 days a week. No gross changes in appearance or condition were observed in any of the guinea pigs during exposure as compared to control animals exposed to clean air. Growth rates of the animals exposed to raw exhaust were not different from the clean air group. Unfortunately, only animals exposed to irradiated diesel (IE) exhaust were evaluated for pulmonary function and ECG patterns. In this latter group, pulmonary flow resistance increased significantly

TABLE 131

Strain A Mouse Data for Induction of Adenomas by Environmental Mixtures

Group	Sex	Percent of Mice with Tumors	Av. Number of Tumors per Mouse	Percent Surviving	Dose
Uninjected	M	40	0.6 ± 0.2	75	--
Controls	F	47	0.6 ± 0.2	85	--
DMSO +	M	40	0.9 ± 0.5	50	0.05 ml/injection
5% EL620	F	47	0.7 ± 0.2	75	
Urethane	M	100^{A}	22.5 ± 1.9^{A}	75	20 mg/mouse
	F	100^{A}	21.8 ± 1.5^{A}	85	
Nissan Diesel	M	40	0.4 ± 0.1	33	4 mg/injection
Particulate	F	20	0.5 ± 0.1	33	
Nissan Diesel	M	73	1.4 ± 0.3^{B}	50	1 mg/injection
Extract	F	55	1.0 ± 0.3	67	
Olds. Diesel	M	37	0.4 ± 0.1	63	1 mg/injection
Extract	F	--	--	--	
Cigarette Smoke	M	59	0.8 ± 0.3	73	0.20 mg/injection
Condensate	F	54	1.1 ± 0.2	80	
Coke Oven	M	54	1.2 ± 0.3	87	0.02 mg/injection
	F	31	0.5 ± 0.2	87	
Roofing Tar	M	41	0.7 ± 0.2	73	0.02 mg/injection
	F	44	0.7 ± 0.3	47	
Experiment 1					
Injected	M	13	0.2 ± 0.1	100	--
Controls	F	26	0.4 ± 0.2	100	--
DMSO +	M	44	0.5 ± 0.2	80	0.05 ml/injection
5% EL620	F	26	0.3 ± 0.1	95	
Urethane	M	95^{A}	7.3 ± 0.7^{A}	100	10 mg/mouse
	F	100^{A}	11.3 ± 0.9^{A}	96	
Nissan Diesel	M	27	0.3 ± 0.1	68	2 mg/injection
Particulate	F	32	0.3 ± 0.1	42	
Nissan Diesel	M	33	0.4 ± 0.1	60	1 mg/injection
Extract	F	50	0.7 ± 0.1	71	
Olds. Diesel	M	33	0.4 ± 0.1	69	1 mg/injection
Extract	F	--	--	--	
Cigarette Smoke	M	30	0.4 ± 0.1	77	0.20 mg/injection
Condensate	F	21	0.2 ± 0.1	80	
Coke Oven	M	31	0.4 ± 0.1	83	0.02 mg/injection
	F	65	0.9 ± 0.2^{A}	89	
Roofing Tar	M	44	0.7 ± 0.2	91	0.02 mg/injection
	F	22	0.3 ± 0.1	91	
Experiment 2					

[A] Significantly different from uninjected and injected controls ($p>0.05$)

[B] Significantly different from uninjected controls and Olds. diesel extract ($p>0.05$)

TABLE 132

Concentrations of Measured Exhaust Constituents Inside Exposure Chambers

	Raw Exhaust
Carbon dioxide (%)	0.26 ± 0.01
Carbon monoxide (ppm)	17.4 ± 2.5
Total hydrocarbons (ppm - C)	31.6 ± 2.3
Nitric oxide (ppm)	5.9 ± 0.6
Nitrogen dioxide (ppm)	2.3 ± 0.4
Sulfur dioxide (ppm)	2.1 ± 0.8
Ozone (ppm)	0
Particulate	
Mass (mg/m^3)	6.32 ± 1.31
Sulfate (mg/m^3)	0.57 ± 0.12

* Values are means ± SD

(Source: Reference 387)

compared to the control animals. However, values for dynamic compliance, minute volume, respiration rate and tidal volume showed no differences at any time between the control and the irradiated exhaust exposed animals. No abnormalities in ECG were noted in any animals although guinea pigs exposed to IE developed a small, but significant, sinus bradycardia.

There was a small but significant increase in the lung weights of animals exposed to both raw exhaust and irradiated exhaust compared to clean air exposed animals being 0.82, 0.78 and 0.68 percent of the body weight, respectively. There were no differences between any of the groups in heart weights. The lungs from both the exhaust-exposed groups of animals showed grey to black discolouration with large deposits of black diesel particulate being present which the authors thought may have accounted for the increased lung weights.

As well, diesel-exposed guinea pigs had increased accumulation of black-pigmented macrophages in their pulmonary alveoli. Granules of black pigment were also noted in focal areas of the tracheobronchial tree and in draining bronchial and carinal lymph nodes. Hypertrophy of goblet cells may have represented a response to pulmonary irritation from diesel exhaust.

Pepelko and colleagues (357) used the Strain A mouse as a model to assess the carcinogenic risk of exposure to diesel engine emissions. This strain of mouse was selected because it has been one of the most extensively used models for assessment of lung tumour induction and the exposure time for development of pulmonary tumours is relatively short. Mice were exposed to 8 hours per day, 7 days per week from 6 weeks to either 9 or 12 months of age at a particulate exhaust concentration of 12 mg/m^3 which was generated from a Nissan 6-cylinder, 90 cu inch diesel engine. Positive urethane and appropriate control groups were included in the study.

Communications with Pepelko of the EPA and Moorman of OISHA provided additional details of studies which are not complete on the effects of diesel exhaust and coal dust on pulmonary function in various species.

In a study in Cyanomologous monkeys in which animals are being exposed to 2 mg/m^3 of coal dust or 2 mg/m^3 of diesel particulate containing known amounts of other diesel emission components, or a combination of 1 mg of coal dust plus 1 mg/m^3 of diesel particulate with the same gaseous contaminant concentrations as in diesel exhaust alone. The exposure is to be for 2 years, 7 hours per day, 5 days per week basis (488). The study is to emphasize pulmonary function, pathology and tumour incidence, as well as immune and mutagenic responses following the exposures.

In addition to the monkey studies, the same group are running similar investigations using Fischer 344 rats in a cancer bioassay and mice and rats in special studies to assess immune potential, mutagenicity and susceptibility to infectivity. The report of this timely and most important series of investigations is to be in the fall of 1983. Preliminary data have been provided by Green et al in a report from the NIOSH Pathology Section, Morgantown, WV. It should be noted that the diesel emissions were generated by a 4-cylinder, 4-

cycle Caterpillar heavy-duty engine, identical to those used in some U.S. underground coal mines. Number 2 diesel fuel with less than 0.5% sulphur was used and the engine was operated in a repetitive 8-mode simulated duty cycle where engine load and speed were varied, with about 55% of the cycle being in the idle mode. Total respirable particulate exposure was maintained at 2 mg/m^3 in all exposure groups, in keeping with current Federal Permissible Limits of Respirable Particulate in Underground Coal Mines. Table 133 shows average concentrations of particulate and gaseous components to which the animals are being exposed. All of the gaseous components are within the permissible exposure limits for airborne contaminants in U.S. mines.

TABLE 133

Chamber Data - 12 Month Summary for Rats

Exposure	CO_2%	CO ppm	NO ppm	NO_2 ppm	SO_2 ppm	NH_3 ppm	Respirable Particulate mg/m^3
Diesel Exhaust	0.21	12.7	9.7	1.6	0.83	1.13	2.01
Diesel Exhaust and Coal Dust	0.20	11.1	8.4	1.3	0.56	0.54	2.08
Coal Dust	0.09	2.4	0.08	0.04	--	0.83	1.97
Filtered Air	0.08	2.3	0.07	0.04	0.01	0.63	--

(Source: Reference 488)

After 12 months of exposure, no significant differences were seen between the four rat study groups for total body weight or for the weights of lungs, heart, liver, spleen, kidney and testes. No differences in hematological indices were observed and no differences were noted in total proteins, albumin, glucose, calcium, phosphorous, cholesterol, uric acid, creatinine, bilirubin, alkaline phosphatase, LDH or SGOT levels. Urine samples collected from male animals exposed for 3 and 6 months were subjected to mutagenic analysis. None of the urine samples from animals exposed to coal dust, diesel emission or coal dust plus diesel emission showed any mutagenic activity, using the Ames Salmonella assay. No changes in the levels of

NADPH-cytochrome C reductase, benzo(a)pyrene hydroxylase or 7-ethoxycoumarin dethylase activities were observed in the liver or lung microsomes of animals examined after 3 months of exposure.

Pulmonary function was measured in groups of rats exposed for 12 months. No significant differences were detected between the treatment groups for any of the variables measured, which included such indices as functional residual capacity, inspiratory and total airway resistance, inspiratory and total airway specific conductance, tidal volume and minute ventilation, etc. Exposure to coal dust, diesel exhaust or a combination of both did not affect the pulmonary tidal ventilatory function of the rats. After pulmonary function testing, the lungs were examined histopathologically. No significant differences were noted between groups as to airspace volume or surface area, or in the development of fibrotic lung disease. There was no evidence of any overt inflammatory process and no lesions involving the respiratory bronchioles.

Sections from animals exposed to coal dust, diesel exhaust and the combination of both all showed similar pathologic changes, which included collections of macrophages containing black, pigmented particles scattered throughout the lungs. The majority of the deposits were within the alveoli, with lesser numbers being found in the connective tissues of the bronchopulmonary tree and within the bronchiolymphoid tissue. Some pigmented macrophages were also found in alveoli adjacent to some respiratory bronchioles. The lining epithelia of these alveoli showed mild cuboidal metaplasia. After three months of exposure, there was no evidence of fibrosis, emphysema or goblet cell metaplasia of the bronchiolar epithelium.

After 6 and 12 months exposure the deposits of pigmented macrophages in the alveoli adjacent to the respiratory bronchioles were larger and resembled coal macules, but there was no evidence of focal emphysema. Tests reflecting changes in elastic lung properties in central and peripheral airways resistance showed no differences between control and exposed animals at 12 months.

It was concluded that there were very few pathologic changes of significance in animals exposed to "low" concentrations of coal dust, diesel exhaust or combinations for up to 12 months. Further evaluation will take place after 24 months of exposure.

In an interim report, published in the Proceedings of the 1982 Diesel Exhaust Symposium, Moorman et al (489) reported on the results of exposure of male cats to 11.7 mg/m^3 of particulate containing 4.37 ppm of NO_2, 5.03 ppm SO_2 and 33.3 ppm CO. Following the first year of exposure to 6.4 mg/m^3 of diesel exhaust particulate, no significant differences were found in mechanical properties, diffusing capacity, uniformity of distribution or ventilatory performance. However, after two years of exposure during which the particulate level was raised to 11.7 mg/m^3, clearly defined pulmonary function responses were reported. Evidence of restrictive disease was found, as demonstrated by a reduction in inspiratory capacity, vital capacity and total lung volume with normal values for ventilatory functions. Thus, there appeared to be restrictive lung disease compatible with pulmonary fibrosis of the interstitial or intra-alveolar type. Additional support for the diagnosis of interstitial disease was the finding of impaired diffusing capacity. Further data on this important study is in preparation.

Although of peripheral interest, the work of Moorman et al (490) on the effects of exposure of monkeys to bituminous coal dust is of some interest, especially as regards particulate exposures and its consequences.

Monkeys were exposed to 2 mg/m^3 of respirable coal dust for 6 - 6.5 hours per day for 24 months. Coal dust was obtained from two sources, including a Pennsylvania coal mine where there was a high prevalence of CWP and a Utah coal mine where there was a low incidence of pneumoconiosis. Pulmonary function was evaluated using an air-tight plethysmographic chamber with the measurement of total lung capacity, vital capacity, inspiratory capacity, residual volume divided by total lung capacity, forced expiratory volume in 0.5 seconds, forced expiratory volume in 1 second, peak expiratory flow, maximum mid-expiratory flow, etc. In addition to the 23 monkeys exposed to coal dust, 10 control animals were carried through the study with exposure only to filtered, clean air. The most significant change in pulmonary function in the animals exposed to either coal dust treatment and the control monkeys was in the MEFV curves, which indicated the development of small airway obstruction. There were significant reductions in V_{max} 40% and 40% TLC. These changes were considered to be consistent with early findings reported in simple CWP. No pulmonary indices discriminated quantitatively between the two coal dust treatments. No restrictive defects were present and normal air flow was found in the larger airways.

4.6 Polynuclear Aromatic Hydrocarbons (PNA's)

As was noted earlier, because of the association between PNA's and diesel particulate, it is difficult to separate the two when considering their toxicity and associated health implications. Therefore, overlap occurs in the discussion in this section and those in 4.5. An attempt has been made to discuss only reports of the effects of PNA's administered alone.

Emminger (358) showed that a single subcutaneous dose of various PAH administered individually, or in combination with other PAH's, in the neck area of mice produced malignant mesenchymal neoplasms at the injection site. The morphology of these tumours suggested that they were similar to sarcomas of the subcutaneous tissue observed in other animal models. The authors suggested that the results strongly indicated that PAH induced mainly neoplasms at the site of their highest concentration.

Pfeiffer (359) administered six different doses of BaP (3 - 100 mcg) and dibenz(a,H)anthracene (2 - 75 mcg) alone and as mixtures subcutaneously to female mice. Mixtures of 10 non-cancerogenic hydrocarbons were used, including benzo(e)pyrene, benz(a)anthracene, phenanthrene, anthracene, etc. It was found that small doses of dibenz(a,H)anthracene had a stronger tumour-forming effect than comparable doses of BaP. With rising doses, the effect of BaP increased more than that of dibenzanthracene. The 10 so-called non-cancerogenic hydrocarbons tested did not produce tumours at the doses selected for this study. As well, the addition of any of the 10 hydrocarbons to benzanthracene or BaP did not affect the dose relationship of the two carcinogenic materials.

Schmahl and co-workers (360) investigated the effect of epicutaneous application of PAH's to mice in stimulating tumor formation. The doses of PAH's used were related to the proportion of the various PAH's found in exhaust gases, as shown in Table 134. It was decided that 87% of the action of the whole mixture of exhaust condensate was attributable to the action of PAH with carcinogenic properties and non-carcinogenic PAH compounds. At very high doses, almost 10 times higher than for carcinogenic PAH's, the group of substances which have been considered to be non-carcinogenic were also biologically active. It was found that the mixture of PAH's was more effective in promoting tumor growth than BaP alone.

TABLE 134

Relative Proportions by Weight of PAH in Exhaust Gas Condensate in Relation to Benzo(a)pyrene

Carcinogenic PAH	
Benzo(a)pyrene	1.0
Dibenz(a,h)anthracene	0.7
Benzo(a)anthracene	1.4
Benzo(b)fluoranthene	0.9
Non-carcinogenic PAH	
Phenanthrene	27.0
Anthracene	8.5
Fluoranthene	10.8
Pyrene	13.8
Chrysene	1.2
Benzo(e)pyrene	0.6
Benzo(ghi)perylene	3.1

(Source: Reference 360)

Pfeiffer (361), in a study of the carcinogenicity of the subcutaneous application to female mice of various polycyclic aromatic hydrocarbons found that exhaust condensates containing BaP were less carcinogenic than pure BaP. It was thought that this inhibition may have been due to adsorption of PAH onto metal compounds or particles in the exhaust condensate. It was also found that small doses of dibenzo(a,h)anthracene had a greater tumor-promoting effect than did comparable doses of BaP. A mixture of BaP and dibenzo(a,h)anthracene was 1.4 times more active than the anthracene compound alone.

Ketkar and co-workers (362) investigated the effect of chronic intratracheal administration of BaP in bovine serum albumin at dose levels of 0.1, 0.33 and 1.0 mg to Syrian golden hamsters. Survival time and incidence data are reproduced in Table 135. Although survival time exhibited dose-dependency, tumor incidence did not. It was found that all dose levels induced papillomas and carcinomas of the trachea, stem bronchi and bronchioles. Interestingly, the incidence of respiratory tract tumours was higher in female hamsters than in males.

In a later study, Ketkar et al (363) examined the influence of low doses (0.125, 0.25, 0.5 and 1.0 mg) of BaP in a mixture of Tris buffer and physiological saline when administered once weekly for life to Syrian golden hamsters. Papillary polyps, squamous cell papillomas and carcinomas developed in both the larynx and trachea. In addition, bronchiogenic adenomas, adenocarcinomas and squamous cell carcinomas were induced in the lung. The highest incidence of respiratory tract tumors (83%) was seen in hamsters receiving 0.25 mg of BaP. The data suggested that doses higher than 0.25 mg, when used chronically, were toxic rather than carcinogenic. A clear dose response relationship for survival time was established as can be seen in Table 136. Multiple instillations of BaP in the saline-Tris buffer mixture resulted in a higher incidence of tumor formation than when the compound was adminstered in either saline or Tris buffer alone, but was still not as high a frequency as when administered in bovine serum albumin. The authors suggested that earlier studies, together with the present investigation, seemed to indicate that particles such as ferric oxide, used as vehicles or carriers do not play an important role in BaP carcinogenesis in the respiratory tract.

As PNA's are attached to particulate matter, additional data relevant to the health effects of PNA's can be found in Section 4.5.

Shabad (364) discussed the establishment of maximum allowable environmental doses for BaP in the USSR, where BaP is regarded as an indicator of environmental pollution with other hydrocarbons, both carcinogenic and non-carcinogenic. The establishment of MAC and TLV values was based mainly on the intra-tracheal instillation of carcinogenic hydrocarbons mixed with adsorbing materials in the induction of lung cancer in rats. It has been calculated that the inhaled BaP safe dose for man is 4.3

TABLE 135

Survival Time and Tumour Incidence in Respiratory Tract of Syrian Golden Hamsters After Multiple Instillations of B(a)P in Bovine Serum Albumin

Group	Effective No. of animals and sex	Average body wt (g) ± SD	B(a)P in mg (solvent)	Survival in weeks ± SD	Respiratory Tract Neoplasms: Incidence No.	Incidence (%)	Trachea No.	Trachea (%)	Lung No.	Lung (%)
1	29 male	115.4 ± 7.0	0.00 (bovine serum albumin)	41 ± 21	--	--	--	--	--	--
	30 female	127.7 ± 13.0		35 ± 20	--	--	--	--	--	--
2	26 male	120.5 ± 7.0	0.10 (bovine serum albumin)	40 ± 20	5	19	--	--	5	19
	30 female	131.4 ± 12.9		34 ± 18	12	40	2	7	10	36
3	29 male	110.1 ± 5.2	0.33 (bovine serum albumin)	24 ± 13	7	24	5	18	2	7
	28 female	127.4 ± 9.7		28 ± 9	10	36	7	27	6	22
4	27 male	91.5 ± 5.7	1.00 (bovine serum albumin)	10 ± 7	6	22	5	19	2	7
	30 female	117.0 ± 6.0		154 ± 11	6	20	3	10	6	20

(SD) - standard deviation

(Source: Reference 362)

TABLE 136

Survival Time, Body Wt. and Tumour Incidence in the Respiratory Tract of Syrian Golden Hamsters After Multiple Intratracheal Instillations of B(a)P in a Mixture of Tris Buffer and Physiological Saline

Group	Effective No. of animals	B(a)P dose in mg	Survival in weeks ± SD	Average body wt. (g) at death	Respiratory Tract Neoplasma				Tumours of other sites
					Incidence No. (%)	Larynx No. (%)	Trachea No. (%)	Lung No. (%)	
1	29 male	Untreated	88 ± 22	116 ± 10	--	--	--	--	1B, 1C, 1E
2	28 male	Vehicle	78 ± 25	114 ± 6	--	--	--	--	1A, 1C
3	29 male	0.125	58 ± 32	127 ± 4	9 (31)	2 (7)	2 (7)	9 (31)	--
4	29 male	0.25	57 ± 22	128 ± 9	24 (83)	2 (7)	13 (45)	21 (72)	1F, 1D, 1G
5	29 male	0.50	28 ± 12	107 ± 7	19 (66)	3 (10)	16 (55)	9 (31)	1H
6	29 male	1.00	14 ± 6	93 ± 8	9 (31)	2 (7)	6 (21)	5 (17)	

SD, standard deviaton; A, thyroid adenoma; B, thyroid medullary carcinomas; C, pancreas duct adenocarcinoma; H, biliary adenocarcinoma; E, hepatocellular adenoma; F, adrenal cortical adenoma; G, squamous carcinoma of the nasal cavity; H, squamous carcinoma of the esophagus.

(Source: Reference 363)

mg during a lifespan, thus concentrations in the air must not be higher than 0.01 mcg/100 m^3. With similar experiments performed using peroral instillation of BaP into mice it was calculated that the BaP content of water should not exceed 0.0003 mcg/l. It was noted, however, that other investigators suggested values up to 70 times higher. Based on epidemiological and toxicological studies, the USSR legislated a BaP limit of 14 mcg/100 m^3 in the workplace air and 0.1 mcg/100 m^3 as the MAC for BaP in atmospheric air.

Further discussion of the potential adverse health effects of PNA is not necessary as their significance was dealt with in the preceeding section (4.5) in association with particulate matter.

4.7 Nitrosamines

Nitrosamines are formed by the reaction of secondary (and occasionally tertiary) amines with nitrous acid in the presence of markedly elevated temperatures and an acid catalyst. These compounds occur widely in nature, as in cereal grains (184, 186). They are also present in low concentrations, less than 5 ppm, in tobacco smoke and alcoholic beverages. These low concentrations have not been assessed for their significance to human health (184).

In the 1950's dimethylnitrosamine was found to be toxic to animal liver. Rats on a diet containing 50 ppm diemthylnitrosamine developed malignant liver tumours in less than a year (365).

In the 1960's, higher concentrations (greater than 5 ppm) of nitrosamines were found in preserved fishmeal intended for animal feeds and this foodstuff proved to be highly toxic to cows and horses (184).

Since nitrosamine formation takes place in the presence of an acid pH, scientists have voiced their concern that toxic or carcinogenic nitrosamines could form in the mammalian stomach. This type of nitrosation was successfully demonstrated in vivo in gastric juice. Recently it has been shown that nitrosamines can be found in the stomach of humans. However, replicative studies are required before firm conclusions can be made. It is known that nitrates (184) are widely distributed in nature, that nitrites are readily formed from them, and it seems likely that nitrite ions are not infrequently present in the stomach of man.

Of the many nitrosamines present in the human environment, a number have proven to be carcinogens or co-carcinogens in many animal models.

Nitrosodimethylamine (DMN) was found to be carcinogenic to all 7 animal species tested by the World Health Organization (366), with the primary target organs being the liver and kidney. The compound induced tumours when given orally, by inhalation or subcutaneously as well as by other routes of administration, in multiple-dose and single-dose experiments. Similarities in the metabolism of DMN by human and rat liver tissues have been reported.

It has been claimed that nitrosamines are formed in the stomach from ingested amines and nitrosating agents such as nitrite. In one dose-response study, rats were fed nitrite and amines in concentrations ranging from 2 - 50 ppm in their diet. At 2 ppm, 4% of the animals developed tumours, at 5 ppm 11%, and at 20 and 50 ppm, liver tumours were observed in more than 66% of the animals. It was considered that a concentration of 5 ppm daily in the diet for a year could be regarded as carcinogenic, which corresponded to a total intake of 54 mg per animal (366).

Although the compound is considered carcinogenic for humans on the basis of animal studies, no long-term follow-up work of human subjects exposed to DMN are known.

Data on N-nitrosodiethylamine (DEN) indicates that it is carcinogenic in all 10 animal specie tested, including sub-human primates. The main target organs are the nasal cavity, trachea, lung, esophagus and liver. It induced tumours following different routes of administration, including ingestion, inhalation and skin painting, and was carcinogenic in single-dose experiments and following pre-natal exposure.

Although much data on the occurrence of DEN in the human environment has been obtained, the analytical methods used were inadequate. The possibility of the in vivo formation of DEN from diethylamine and nitrosating agents needs further attention. No long-term studies of human subjects exposed to DEN were found (366).

The limiting factor in the formation of nitrosamines would appear to be the availability of nitrosatable secondary amines. These compounds occur in fishmeal and fish products, cereals, tea and in tobacco and tobacco smoke.

Two amines which occur in these ingestibles and inhalants are dimethylamine and diethylamine. Although conversions of either of these amines to nitrosamines is not favoured by the generally dilute acid conditions of the stomach, due to the relatively strong basic nature of these amines (184), the assumed frequency of nitrosamines in the human environment means there is an increased opportunity for nitrosation to take place.

Cooking and food preservatives offer sources of secondary amines. The possibility exists that proline, ingested in food, can be converted into nitrosoproline by nitrite present in the stomach. Subsequent chemical reactions could yield the highly-carcinogenic nitrosopyrrolidine.

Cigarette smoke contains many secondary amines, including pyrrolidine and piperidine. These amines could dissolve in the saliva during smoking and be converted to nitrosamine in the stomach. Such nitrosamines may be absorbed and give rise to tumours of the lung, a property of several nitrosamines (184).

The formation of DEN from inactive precursors, diethylamine and nitrosating agents has been investigated (366). DEN formation was reported as having occurred in gastric juice in the presence of sodium nitrite and a few crystals of diethylamine hydrochloride. The formation of DEN from diethylamine and methylnitrite, both present in tobacco smoke, has been demonstrated. The formation of nitrosamines has also been shown under neutral conditions from secondary amines and nitrate in the presence of certain bacteria present in the human digestive tract or in urinary tract infections.

Nitrosomethylurea and nitrosoethylurea have both been found to be carcinogenic in several animal species. In addition, there are data to indicate that nitrosomethylurea may be formed in vivo in man from a combination of methylurea and nitrite, or other nitrosating agents. Similarly, nitrosoethylurea may be formed from ethylurea by nitrosation in vivo (366).

Studies by Sander's group indicated that only readily-nitrosatable compounds were sufficiently nitrosated in vivo for tumours to be induced. It was noted that when using a particular strain of mice, NO compounds induced many lung adenomas per mouse which could easily be counted. The adenomas appeared to arise independently and seldom metastasized. Experiments with preformed NO compounds showed that the yield of adenomas was nearly proportional to the nitrosamine dose. However, the probability for these compounds to be formed in vivo was not great, and the system for formation was inefficient since food and drinking water containing nitrites must be present in the stomach simultaneously for nitrosation to occur. The compounds given separately had no effect. For intragastric nitrosation to be important, nitrite must not disappear rapidly from the stomach. All these processes probably require conversion of nitrite to HNO_2 by gastric acid. Formation of nitrosamines by in vitro incubation of secondary amines and nitrite in the gastric juice of various species, including man, has been demonstrated (186).

The effect of DEN in producing tumours of the trachea and of the lung in hamsters has been studied. Doses were 1 mg intragastrically, subcutaneously or by inhalation, once or twice a week for 1 - 5 months.

A dose of 0.75 mg of DEN per animal was found to be the smallest single dose causing papillary tumours in the adult hamster trachea 25 weeks after treatment. Smaller doses were capable of inducing tumours after longer times.

Observations of lung tumour development in man do not always correlate with early alterations seen in the hamster model. It is known that in man, metaplasia of the bronchial epithelium, mainly after chronic inflammation, is not necessarily connected with the histogenesis of bronchogenic carcinomas (367).

It has been demonstrated that a causal relationship exists betwen cigarette smoking and the incidence of lung cancer, but there is relatively little information as to the characterization of the carcinogen responsible (368).

Other workers have shown that benzpyrene present in tobacco smoke can account for no more than 1% of the carcinogenic properties of cigarettes.

Evidence indicates that at least three nitrosamines may be present in cigarette smoke. No reliable quantitative determination of the amount of nitrosamines present could be obtained, but a rough estimate predicts that approximately 1 - 5 ug of N-nitrosopiperidine is formed per cigarette.

It should be noted that the nitrate content of South African cigarettes was usually much higher (1 - 2%) than the values normally accepted in other countries (368).

A significant incidence of pulmonary tumours has been reported in both rats and mice as a result of the simultaneous administration of 3-methylcholanthrene and dimethylnitrosamine at levels at which neither the nitrosamine nor the hydrocarbon alone were carcinogenic to the lung.

Since nitrosamines, including dimethylnitrosamine, and hydrocarbon (3,4-benzopyrene and 3,4-benzofluoranthene) are very similar in carcinogenic properties to 3-methylcholanthrene, which is present in tobacco smoke, it is proposed that a substantial proportion of the lung tumour incidence of smokers may be due to a synergism between the carcinogenic hydrocarbons and nitrosamines present in the smoke, rather than to the hydrocarbons alone. Two alternative mechanisms are under consideration with regard to the methylcholanthrene-nitrosamine synergism. First, demonstration of synergism between particular carcinogenic hydrocarbons and nitrosamines present in tobacco smoke could open unexplored approaches toward partial protection of smokers, and second, modification of a diet, rich in nitrosamine contaminants or pre-nitrosamine components, might play a role in lung tumour reduction in smokers (369).

Nitrosation has been shown to occur on occasion in the human stomach, but it cannot be shown to occur in the lungs. However, further study is required, along with detailed analysis of nitrosamines in cigarette smoke, as approximately 32 to 50% of underground miners are moderate smokers.

At the present time, too little is known to establish guidelines for the control of nitrites as materials for nitrosation and no atmospheric levels can be set or discussed with reference to health effects until their presence in the mining atmosphere is better defined.

Gross and Newberne (370) provided an excellent summary review of the nitrosamine-carcinogenicity problem, including a discussion of the evidence which exists for the occurrence of the in vivo formation of nitrosamines. They emphasized that despite strong evidence of wide-spread potential for human exposure to nitrosamines, there is no direct evidence linking their carcinogenicity in man. As was pointed out above, no information is available which would suggest that nitrosamines are formed in lung tissue, although there is a considerable body of information demonstrating their formation in the gut. They also called attention to work completed in late 1960 which showed a synergistic effect of nitrosamines and polycyclic hydrocarbons in inducing respiratory tumours. However, as there is no epidemiologic evidence to suggest an increased rate of respiratory carcinogenesis in miners exposed to diesel exhaust, this would not appear to be of practical importance.

4.8 Acid Mists

Sulphur oxides emitted by diesel engines can give rise to sulphuric acid, particularly if the engine is equipped with a platinum catalytic purifier through oxidation of a portion of the SO_2 emitted by the engine.

Acid mist levels in the Denison Mine were studied by Kirk and Stefanich (121) in an area with a high concentration of diesel exhaust. The area of sampling was considered to be a "worst possible case" situation and was not representative of the entire mine. Respirable and total dust samples were collected and a specific ion electrode test for sulphuric acid was carried out.

The fuel being used was Grade 1A with a sulphur content of 0.21%. The concentrations of dust and sulphuric acid measured are given in Table 137. These results indicated that there are significant quantities of sulphuric acid present around diesel equipment. Since the acid concentration was the same for both the respirable fraction and the total dust, the acid was probably adhering to diesel soot particulate. Theoretical calculations indicated that the sulphuric acid content could go as high as 0.39 mg/m^3 for a diesel fuel containing 0.5% sulphur. Interestingly, of the respirable dust in the Denison Mine, 80% was combustible, and this would contain the soot hydrocarbon derived from diesel emissions (121).

There is only limited published information on the effect of sulphuric acid on the respiratory tract and on pulmonary function in human subjects. In 1971, one study described human subjects who were exposed to sulphuric acid mist having a mass median diameter (MMD) of 1 u. The concentration ranged from 0.35 to 5 mg/m^3 and exposure times were from 5 - 15 minutes. Respiration rate, tidal volume and minute volume were measured. In 15 subjects exposed to 0.35 - 0.5 mg/m^3, the respiration rate increased about 30% above control values. The maximum expiratory flow rate decreased by about 20% and tidal volume increased about 28%. The changes occurred during the first 3 minutes of exposure and were maintained throughout the 15 minutes of exposure. When the exposure ended, the values returned to pre-exposure levels within 15 minutes.

TABLE 137

Summary of Results

	Respirable Dust	Total Dust
Average dust concentration (mg/m^3)	0.73 ± 0.06	1.07 ± 0.09
Average H_2SO_4 (mg/m^3)	0.17 ± 0.01	0.17 ± 0.04
Percent H_2SO_4	23 ± 3	16 ± 2

(Source: Reference 121)

At concentrations of 5 mg/m^3, the acid mist was perceptible to all subjects. This study did not establish that obstructive pathology was the result of sulphuric acid exposure, but it was suggested by the pulmonary function measurements (371).

Exposure of young animals to 0.9 micron (u) of sulphuric acid produced the following pathological picture: the lungs were slightly edematous and a rare capillary hemorrhage was noted. Major and minor bronchi showed a reduced amount of mucous and less spasm in exposed animals than in controls. It was concluded that the guinea pig could tolerate about 2 mg/m^3 of sulphuric acid for more than 3 months of continuous exposure with only minor effects (372).

The first experiment aimed at determining the interactions of individual and combined oxides of sulphur, SO_2 and H_2SO_4, in normal animals compared to those that had previously been exposed to NO_2 and had lung pathology. Residual functional effects were determined in these dogs. The second part of the study examined the pulmonary function in dogs exposed to NO_2 plus a relatively inert dust, iron oxide. Dogs were chronically treated with 26 ppm NO_2 for 6 months. Concentrations of the other components used were 5.1 ppm SO_2, 755 ± 362 ug H_2SO_4 and 922 ± 535 ug iron oxide.

Dogs that had received the chronic exposure to NO_2 showed no significant change in pulmonary function. It can be concluded that if there were destructive changes in the lungs, they were not sufficiently severe to result in significant physiological abnormality. Sulphuric acid decreased the diffusion capacity of all treated groups. The reason for this is difficult to interpret without histopathological data (420).

A study of lung tissues was undertaken in guinea pigs (372). Exposure times were extended for 72 hours at a concentration of 8 mg/m^3. The pathological examination of the guinea pig lungs showed much greater damage from the longer exposures. Thickening of the alveolar walls and areas of consolidation were observed. The lung damage of animals exposed to the high concentration for 72 hours was much greater than in animals exposed for 8 hours. It was postulated that sulphuric acid mist had two distinct toxic actions: to promote laryngeal spasm and spasmotic bronchostenosis, which is the cause of death; and to produce parenchymal lung damage. This latter effect is probably a direct irritant action of the acid mist deposited on the lung tissue.

In a summary of other investigators' experiments, the results of guinea pig exposure of up to 4 mg/m^3 sulphuric acid mist for 18 - 140 days, of particle sizes 0.6, 0.9 and 9 microns were discussed. Sulphuric acid mists consisting of 0.9 u particles produced greater lung damage than either the 0.6 or 9 micron particles (372).

Amdur reviewed the data of Sim and Pattle in which healthy males were exposed to 10 minutes by mask and 60 minutes by chamber to a sulphuric acid mist at a relative humidity of 62% and a droplet size of 1 micron. The sulphuric acid was found to be more irritant under conditions of high humidity. These researchers concluded that there was no question that human subjects, as with guinea pigs, had a greater irritant sensitivity to sulphuric acid than to an equivalent amount of sulphur as SO_2 (372).

Amdur commented that many studies in the literature used standard exposure times of one hour and therefore had no relevance to the long-term effects of sulphuric acid mists. The results did, however, indicate that the particle size of the aerosol was a critical factor in determining both the nature and degree of irritant response.

Seven micron particles, at a concentration of 30 mg/m^3, produced an increase in flow resistance, but no other detectable changes in respiration. Particles of this size would not penetrate beyond the nasal passages.

The 0.8 micron particles produced an increase in resistance, and although to a lesser degree, an increase in the elastic and resistive properties of lung tissue and total work of breathing. These changes were significant at the lowest concentration tested, which was 1.9 mg/m^3. The response was prompt in onset and resembled the pattern of changes observed after inhalation of SO_2.

Comparison of the data shows a difference in the physiological action of two different particle sizes, which was probably related to the site of deposition of the particles in the respiratory system. For example, the response to 2.5 micron particles was slow to develop compared to 0.8 micron particles, whereas at the end of one hour exposure, the response to the larger particles was much greater than to 0.8 micron particles (372, 373).

In summary, the findings suggested that with high concentrations of larger particles which were probably deposited at about the level of the major bronchi, local irritation of the larger air passages caused swelling and increased exudation of fluid, leading eventually to complete obstruction.

Of interest was the finding of a slow return to normal after exposure to sulphuric acid mists suggesting that the particles, having been depsoited on the respiratory tract, continued to exert their irritant effect.

Not only was the response to a given concentration greater as the particle size decreased, but also the dose-response curves were steeper as the particle size decreased. The results emphasized that if a compound was dispersed as particulate material a measure of the mass concentration alone does not permit assessment of its irritant potency.

It was noted that an equivalent amount of sulphur produced a lesser irritant response if it was present as sulphur dioxide gas rather than as particulate sulphate or sulphuric acid. One ppm of SO_2 is the equivalent of 1.3 mg of $sulphur/m^3$; when present as SO_2 gas, this amount of sulphur will produce an increase of about 15% in flow resistance. If, through reaction in the atmosphere, the sulphur equivalent was converted to 0.7 micron sulphuric acid, it would produce an increase in resistance of approximatley 60%, a 4-fold potentiation in irritant response.

Considering that a 10% oxidation of SO_2 could readily occur in the atmosphere, it was estimated that from 1 ppm of SO_2 an equivalent sulphur content of 0.13 mg/m^3 would be formed. If present as an irritant sulphate of 0.3 micron particle size, an increase in resistance of approximately 45% would be produced (372).

Schiff and associates (479) studied the effect of sulphuric acid mist (1.1 mg/m^3) administered alone and in combination with carbon particles on ciliary motion and airway histology in animals. A reduction in ciliary beating frequency was recorded with the acid mist alone and with the acid mist-carbon mixture. Immediately after exposure, the acid mist and carbon mixture animals showed significantly greater tracheal epithelial cytological alterations than those exposed to either of the pollutants alone. Three days after exposure, only the combined treatment group showed significant damage compared to controls. The data would suggest that in this model, mixed pollutants were more toxic than single pollutants.

Malcolm and Paul (376) reported on the effect of sulphuric acid on the teeth of workers in the storage battery industry. It was found that those workers who were exposed to acid mists displayed erosion of the incisors. The concentration of acid in the air, as sulphuric acid, varied from 3 to 17 mg/m^3. Men exposed to the latter concentration showed the greatest amount of erosion. In other workplaces where the amount of acid in the atmosphere varied from less than 0.8 to 2.5 mg/m^3, dental erosion was apparent, but not as severe as in the above instance.

Five hundred and fifty-five workers were exposed to acid fumes in their work environment and were examined between March, 1962 and October, 1964 (374). Approximately 32% were affected by dental erosion due to the acid. Hydrochloric acid and sulphuric acid accounted for far more erosion than nitric acid. No quantitative data was provided which would be useful in setting concentration limits for acids that prevent dental erosion.

Sachner and co-workers (375) investigated the safety of brief exposures to sulphuric acid aerosols in high concentrations. Dogs that breathed H_2SO_4 aerosol in concentrations up to 8 mg/m^3 showed no effects on respiratory resistance, static lung compliance or functional residual capacity. A four-hour exposure to 4 mg/m^3 produced no significant changes in mechanics of breathing, functional residual capacity, pulmonary and systemic arterial blood pressures, cardiac output, heart rate or arterial blood-gas tensions. Normal and asthmatic adults breathing H_2SO_4 aerosol in concentrations up to 1 mg/m^3 for 10 minutes showed no significant alteration of lung volumes, distribution of ventilation, dynamic mechanics of breathing, pulmonary capillary blood flow, or other indicators of cardio-pulmonary function. The study demonstrated that single exposures to sub-micronic H_2SO_4 aerosol did not produce an immediate or delayed adverse effect on the cardio-pulmonary function in anesthetized dogs, conscious sheep or normal and asthmatic adults.

4.9 Oil Mists

Only recently has concern arisen over the health effects of fine particles of oil in a mist. The oil comes entirely from hydraulic equipment such as the drillers and bolters, which are usually mounted on diesel-powered units.

Although smooth surfaces underground (i.e. the crown of a safety helmet) pick up a fine palpable layer, very little is written in the journals regarding this aspect of the mine environment.

The mixture of carbon soot and oil that made up the ink-mist aerosol found in high-speed newsprint pressrooms of the New York Times was described by Lippman and Goldstein (377). This mist caused a reduction of visibility during the press run and fallout on exposed surfaces. The New York State Labour Department demonstrated that the gross air concentrations of ink mist in the pressroom was frequently above the ACGIH threshold limit value (TLV) of 5 mg/m^3. This article investigated the problem. The high-speed presses were routinely operated at 55,000 newspapers per hour. The newer presses were designed with a more complete enclosure of the rollers and thus resulting in less fallout. The oil in the ink consists of extract products from lubricating oil refining processes. The carbon black in the ink is made of an amorphous elemental carbon from furnace processes. The particle size of carbon black of the diameter of primary particles was 27 microns, and the size of disperse aggregates was 0.1 u to 0.2 u.

The ink-mist particle size showed that in a total of 36 samples, the average median droplet diameter was about 14 microns and the range was 9 to 30 microns. This type of aerosol is quite unstable because of the rapid sedimentation of the large droplets and it can be maintained only during press operation. The concentration of the after-sample, which began immediately after the end of the press run, was very low. This suggests that the coagulation, coalescence and fallout of the large droplets during the press run tends to scavenge by coalescence the smaller oil-bearing backround dust from the room atmosphere (377).

Time-weighted average ink-mist exposure should be redefined for the gross mass concentration and respirable mass concentration. The pressroom crews were overexposed by 20 - 45% on the basis of gross concentration. This level of exposure would not be expected to produce any measurable health effects. The 5 mg/m^3 TLV was established as a nuisance limit more than a health limit. The TLV should be interpreted as applying to respirable particles. On the basis of respirable mass concentration, the highest time-weighted average exposure of 1.4 mg/m^3 was well below the TLV of 5 mg/m^3 (377).

Other studies show the same postive attitude. One study suggested that oil mists of automobile exhausts could serve as the nuclei for aerosols on which exhaust gases may be adsorbed. Hence, the toxicological properties of the gases would be altered. Also, lipid pneumonia was not a hazard in one study where animals were exposed continuously for one year to levels of oil from 63 ug to 132 ug per litre.

There is no evidence in the literature of relevant studies done related to the health effects of oil mists in mine atmospheres.

4.10 Aldehydes, Acrolein and Phenols

Aldehydes present in diesel exhaust emissions result from incompletely-burned carbonaceous fuels. Aldehydes are irritants and those exposed to elevated levels complain of itching, tearing eyes, as well as sore, dry throats. Other physiological changes are probably due to effects on the upper airways.

The other undesirable effect of aldehydes is the contribution made to the unpleasant odour of diesel exhaust. Two main contributors in this respect are acrolein and formaldehyde which are the most thoroughly investigated compounds. However, recent studies have shown that oxides of nitrogen are more likely to be noticed ahead of aldehydes as odourous elements in diesel exhaust with all these compounds varying with many factors related to diesel engine status.

Humans show some adaptation to formaldehyde up to levels of 20 ppm. However, some individuals appear to become more susceptible on repeated exposure and some are very sensitive to formaldehyde from the very first exposure. The latter suffer from mucosal irritation in atmospheres of 5 to 6 ppm and some workers have suffered symptoms in atmospheric levels as low as 1 to 2 ppm of formaldehyde (23).

Amdur's study in 1960 (378) exposed guinea pigs to formaldehyde at three aerosol concentrations of 3, 10 and 30 mg/m^3. With concentrations as low as 0.3 ppm, there was an increase in airway resistance and a decrease in compliance. As the aerosol concentration increased, the respiratory changes became more marked. The change in pulmonary resistance was reversed one hour after exposure to formaldehyde ended (378).

Acrolein has been found in numerous studies to be the most toxic of the aldehydes at low atmospheric concentrations (23). Levels as low as 1 ppm for 5 to 10 minutes caused eye and respiratory tract irritation. It is of interest to note that acrolein was the most significant of the irritating components in cigarette smoke.

Recent studies exposed mice to low levels of acrolein. In one study, decreased pulmonary compliance was observed. In another, in which mice were exposed for 63 days, body weight changes were recorded as well as diminished liver weight and respiratory irritation.

There is no evidence of chronic effects or permanent respiratory tract changes based on long-term studies of animals or humans who have been exposed to diesel engine exhaust. Further studies of exhaust emissions need to be undertaken to ensure that levels at or below the present TLV of 0.1 ppm acrolein are maintained until more specific animal and human studies are completed.

Phenol and other aromatic compounds, such as benzene, are also products of incompletely-burned fuel found in diesel exhaust. Most of our knowledge derives from observations of workers exposed to phenol vapours in industry.

Levels of 8 to 48 ppm in a single room resulted in the workers suffering marked irritation of the eyes and upper respiratory tract. In another plant area, the level was 4 ppm, where only the phenolic odour was noticed (23).

A mixture of phenol and formaldehyde has been found in diesel exhaust, as well as in many industrial situations such as acrylic plants.

Schoenberg and Mitchell (379) in 1975 examined workers from an acrylic plant who were designated as never-on-line or currently-on-line in terms of exposure. The fume concentrations ranged from less than 5 ppm to 30 ppm of formaldehyde. Phenol was 7 - 10 mg/m^3 and formaldehyde was 0.5 - 1 mg/m^3, on two separate days of the study.

It was found that on-line workers had excess cough and sputum. The differences between groups in cough and cough plus sputum were statistically significant. The on-line workers had a significantly lower FEV_1/FVC ratio than the never-on-line group. The controls had small increases in pulmonary function test results during the study. The on-line workers showed small decreases in all pulmonary function tests on Mondays with decreased FEV_1 and maximum expiratory flow (MEF) 50% during the week (379).

This study suggested that long-term exposure to phenol and formaldehyde resin fumes resulted in chronic airway obstruction, as shown by FEV_1/FVC and MEF 50%/FVC ratios. The differences between on-line and never-on-line workers suggested that the chronic effects of formaldehyde and phenol resin fumes may be reversible. The pulmonary function indices indicated that the presently-on-line workers with longer exposures had more airway obstruction (379).

Morgan (480) reviewed industrial bronchitis in which he emphasized that bronchitis and airway obstruction are not necessarily related. Bronchitis can exist in a simple form with hypersecretion of mucous in the absence of ventilatory impairment and as obstructive bronchitis in which an increase in cough and sputum is accompanied by a reduction in ventilatory function or capacity. Chronic bronchitis is a condition that affects the large airways and is characterized by an excessive amount of mucous. In contrast, it is the peripheral airways, those distal to the 12th generation, that are the anatomic site of the pathologic changes that occur in irreversible airway obstruction. In summarizing the clinical determination and background of chronic and industrial bronchitis, Morgan pointed out that cigarette smoking affected the flows at all lung volumes, that industrial bronchitis predominantly affected peak flow and flow at high lung volumes. He noted that industrial bronchitis was primarily a result of dust exposure. He also pointed out that because of mouth breathing, particularly under exertion, some of the larger dust particles in the 5 - 10 um range will be deposited in the respiratory tract, and he suggested that in future when designing studies with the purpose of relating biologic and environmental measurements that both respirable and total dust should be measured. He concluded that much of the reduction in ventilatory capacity that has been observed in dusty occupations is a reflection of industrial bronchitis rather than parenchymal involvement such as pneumoconiosis. It was pointed out that although industrial bronchitis could be viewed as a cause of ventilatory impairment, its presence could not necessarily be equated with disability. Morgan concluded that non-smokers who worked in dusty occupations may have chronic bronchitis, but as a group will have only minimal impairment of air flow, which is probably due to large airways involvement. In workers who smoke, the effect of smoking as a cause of significant airway obstruction is 5 - 10 times greater than the dust.

Andersen and co-workers (481) evaluated the effect of exposure to an inert polymerized plastic dust at levels of 2, 10 and 25 mg/m^3 for five hours to human volunteers. The main result of this exposure was dryness in the nose and pharynx with some minor changes in pulmonary function.

4.11 Silica and Dust

Quartz, with its varying content of silica, has in the past posed a serious problem for hard rock miners including uranium miners because of the early or late development of silicosis, with its attendant debilitating disease, silico-tuberculosis.

In spite of increasing stringency of mine regulations regarding the control of dust, especially silica dust, a significant number of new cases of silicosis may be expected to appear in the coming decade because of the long latency period for its development. The average lifespan of silicotics in Ontario is approaching the average life expectancy of Canadian males who are the average age at which silicotics are recognized (380).

The opening of Ontario's uranium mines in the 1950's led to a rapidly-developing silicosis in uranium miners. However, the newer, more demanding mine regulations now in effect are expected to lead to a decrease in the incidence of silicosis.

In the Royal Commission's report on mines in Ontario (381), statistics showed that to the end of 1974, no case of silicosis charged to the uranium mines involved a person who first entered dust exposure in Ontario in or after 1960, that is, in the fifteen-year period before 1974.

The konimeter continues to be used to take "grab samples" in the working place, but there are wide variations between samples taken at one location over half an hour intervals. Industry-wide dust control in Ontario mines continues to be based on the konimeter. Such measurements are not indicative of an individual miner's personal exposure to dust over a working shift and hence such measurements are inadequate.

The TLV for respirable free silica as introduced by the ACGIH 1968 is effectively 0.1 mg/m^3 under a specified size-selection characteristic.

Silicosis can develop with exposures to dust with 5% quartz or greater. The literature supports the conclusion that a significant increase in risk can occur with dust with an 18 - 20% quartz content and hence our ventilation recommendations (see Section 6) suggest different exposure limits, depending on the quartz content of the dust.

4.12 Radiation

In the uranium mine, the lung is directly exposed to naturally-occurring radioactive materials through inspired air, as outlined and described in Section 3.6. When considering the underground mining environment, it is important to examine the sources of natural radiation and consider the possible consequences of human exposure to chronic, low-dose radiation from airborne radionuclides. The lung is unique in that it can receive radiation from internally-deposited material, such as radon daughters adherent to particulates, while at the same time being continually exposed to air from the external environment, which could contain radioactive radon gas (382).

Uranium ore usually contains less than 0.5% uranium, but occasionally pockets with 5% or more are found. The most abundant isotope is uranium-238 which decays through a series of solid elements to radium-226, which has a half-life of 1,620 years and decays to the gas radon-222 with a half-life of 3.82 days. It should be noted that in addition to radon-222, three of the radon daughters are alpha-emitters, but only RaA and RaC' are considered to be significant alpha-emitters in evaluating potential harm to the miner (383).

The polonium-218 atom formed by the decay of radon-222 and the other radon daughters exist as a free ion in air for less than a minute. It has been shown that the radon daughters quickly become attached to moisture or dust particles which may be inhaled.

In still air, radon-222 is in equilibrium with its short-lived daughters within 3 hours (that is, the ratios between the amounts of various members of the series remains constant), but in working areas of the mines, equilibrium may not be reached except with RaA, the first daughter produced. The RaA atom formed by the decay of radon-222, and the other radon daughters exist as free ions in air for less than a minute. It has been shown that the radon daughters quickly become attached to moisture or dust particles which may be inhaled or retained in the lungs.

It can be calculated that the major radiation dose to the lungs, where the radon daughters were deposited, is derived from the alpha particles emitted by RaA and RaC', and to a lesser extent, by the alpha particles from radon-222.

The range in tissue of alpha particles produced by radon and its daughters are given in Tables 138 and 139. The heavy alpha particles cause dense ionization along their path in tissue and this is particularly intense when the energy is reduced to less than 2 MeV. Although the initial energy of the radon daughters is greater than 2, by the time they have passed through mucous and cilia and reach the basal cells of the bronchial epithelium the energy would have been reduced to 2 MeV or less. Thus, the nucleus of the basal cell is likely to be subjected to ionization damage. The current assumption is that such damage could result in malignancy (383).

TABLE 138

Range of Radon and Radon Daughter Alpha Particles in Tissue

Nuclide	Energy (MeV)	Range in Tissue (Microns)
Radon 222	5.49	41.1
Radium A	6.00	47.0
Radium C'	7.68	70.8
Radium F	5.30	38.9

(MeV - million electron volt)

(Source: Reference 383)

A clear relationship exists between exposure to airborne radiation expressed as WLM, and lung cancer deaths. With exposures of 840 to 1800 CWLM (cumulative working level months), there was a marked, progressive lung cancer risk. Below 840 WCLM, 25 deaths occurred compared to the 7.2 expected, but most important was the observation that there was an excess of deaths (10 observed against 2.4 expected) among miners receiving 120 to 359 CWLM. The increased death rate could be interpreted as a four-fold risk for miners exposed to 1 working level for 10 - 30 years. Smoking uranium miners experience an excess of lung cancer 10 times greater than non-smoking miners, suggesting a synergistic effect with cigarette smoking.

The work of Saccomanno and others found that in uranium miners with lung cancer, only one of the 150 uranium miners was not a cigarette smoker. Williams pointed out that one can hardly avoid the conclusion that cigarette smoking and inhaled radon daughters are synergistic carcinogens.

Some authorities suggest that there is insufficient data available to arrive at an accurate figure for a "safe" exposure to radon daughters. Moreover, in view of the current assumption for protection purposes that no threshold dose for radiation effects exists, one must select a level of exposure which will not subject a miner to an unreasonable hazard. Holoday and his associates suggested a working level of 1.3×10^5 MeV of potential alpha energy per litre of air for radon daughter products; this has been the standard applicable to the control of radon-222 and its short-lived daughters

TABLE 139: Properties of Radon and Its Daughter Products

Isotope	Atomic weight	Atomic number	Emission	Energy of the particle, MeV	Range of α-particle in the tissue, u	Half-life period	Disintegration constant, min^{-1}	Biological half-life, days	Disintegration constant, min^{-1}	Corresponding element
Rn	222	86	α -	5.486	41	3.825 days	1.258×10^{-4}	30 min	--	Rn
RaA	218	84	α -	5.998	47	3.05 min	0.2272	100 days	0.2272	Po
RaB	214	82	β -, α -	0.65	--	26.8 min	0.0259	700 days	0.0259	Pb
RaC	214	83	β -, α -	3.15	--	19.7 min	0.0352	3 days	0.0352	Bi
RaC'	214	84	α -	7.680	71	1.64×10^{-4} sec	2.8×10^{-5}	100 days	2.8×10^{8}	Po
RaD	210	82	β -	0.0255	--	22 years	6.0×10^{-8}	700 days	7.4×10^{-7}	Pb
RaE	210	83	β -	1.17	--	5 days	9.6×10^{-8}	3 days	2.6×10^{-5}	Bi
RaF(Po)	210	84	α -	5.298	39	138.374 days	3.438×10^{-6}	100 days	8.3×10^{-6}	Po
RaG	206	82	Stable	--	--	--	--	700 days	--	Pb

Note: Biological half-life period of RaA, RaB, and RaC (RaC'), which enter while breathing, is about 8 hours (119). The average energy of α-particles is equal to 1/3 of the maximum energy for RaB (0.65 MeV) and RaC ($0.23 \times 3.17 + 0.77 \times 1.65 = 2$ MeV).

(Source: Reference 384)

in Canada and other countries. Subsequently, a regulation was put in place reducing the permitted WL by a factor of 3, allowing a maximum of 4 WLM per year and 2 WLM in any three-month period. Such a standard permits a miner to work for 25 years without being subjected to a significantly increased lung cancer risk. Some authors suggest that there is a practical threshold and that a cumulative lifetime exposure of 300 - 400 WLM would carry only a negligible risk of developing lung cancer from radiation (383).

In one study, hamsters were exposed to radon daughters, uranium ore dust and diesel engine exhaust. Each chamber of 102 hamsters was exposed to one or a combination of pollutants, as follows:

	No. Hamsters	Radon Daughters	Uranium Ore Dust	Diesel Engine Exhaust
Group 1	102	--	--	
Group 2	102	1200 WL	--	
Group 3	102	1200 WL	15 mg/m^3	
Group 4	102	--	15 mg/m^3	
Group 5	102	--	--	50 ppm CO
Group 6	102	1200 WL	15 mg/m^3	50 ppm CO

Radon daughter samples were taken daily from the chambers and a silicone diode detector provided simultaneous analyses for RaA and RaC' activity. These measurements were used to control radon daughter exposure in order to obtain mean exposure levels of 1200 WL (385, 386).

Uranium ore dust concentrations in Groups 3 and 4 varied during the last years of exposure, between 15 mg/m^3 and 20 mg/m^3. Particulate aerodynamic mass diameters of 0.3 to 1.1 microns were measured.

Air samples taken in uranium mines as part of integrated AEC studies in the past years (1972 - 1973) showed that the presence of submicron-sized uranium ore and/or diesel exhaust aerosols markedly increased the levels of condensation nuclei to which radon daughters attach, affecting their attachment fraction (especially for RaA) and physical behaviour (385, 386).

The trachea and lungs were analyzed separately in order to observe possible differences in RaB versus RaC levels in the first order airway, as compared to the remainder of the lung. Group 6 hamsters, sacrificed in March of 1973, showed lung RaB and RaC mean radioactivity levels of 4.0

and 6.1 x 10^4 dpm/gram, respectively, and tracheal RaB and RaC mean total radioactivity levels of 0.32 and 0.58 x 10^4 dpm. Corresponding lung mean radioactivity levels for Group 2 hamsters of RaB and RaC were 7.3 and 14.5 x 10^4 dpm/gram, respectively, and mean total RaB and RaC radioactivity levels for trachea were 0.57 and 0.9 x 10^4 dpm.

Mean lung weights for sacrificed Group 6 hamsters and Group 2 hamsters at this time (11 months of exposure) were 1.5 g and 0.91 g, respectively, compared to 0.78 g for controls. These increased lung weights reflected the progressively greater pulmonary congestion of hamsters inhaling diesel engine exhaust and/or ore dust with radon daughters as compared to radon daughters alone. This effect of increasing lung weight, with similar or possibly decreased ventilation, may cause relatively higher lung radiation doses at later times in Group 2 than in Group 6 animals.

Hamsters receiving 30 WL of radon daughters showed a slight pulmonary response, and lifetime exposures to 600 WL radon daughters with ore dust caused pulmonary lesions including emphysema, fibrosis, metaplasia and adenomatous lesions with anaplasia (Phase I studies). Phase II studies involved lifespan exposure to 1200 WL radon daughters, diesel engine exhaust and uranium ore dust, which caused more extensive and severe pulmonary lesions with accelerated development of epithelial cell metaplasia and the appearance of squamous tumours. Earlier studies in another laboratory with mice exposed to 1750 WL radon daughters (150 hours/week) showed no increase above the spontaneous control incidence of adenomatous tumours.

Syrian hamsters, SPF rats and mice were exposed to atmospheres containing 3000 to 6000 WL of radon daughters with normal room air dust, or with 15 - 18 mg/m^3 of uranium ore dust. All three species died earlier when exposed to radon daughters with uranium ore dust than to radon daughters alone (385, 386).

Dogs receiving daily exposure to radon daughters with uranium ore dust, plus cigarette smoke showed total white blood cell counts significantly higher than in the control dogs or other treatment groups. This was due entirely to an absolute neutrophilia. A modest, but significant monocytosis was previously observed in this group exposed to radon daughters with ore dust plus cigarette smoke. The origin of the neutrophilia observed in dogs exposed to radon daughters with uranium dust plus cigarette smoke appeared to be the result of chronic irritation and increased pulmonary cell death from the synergistic effects of the radon daughters, uranium dust and cigarette smoke.

Chest x-ray of Group 2 chests visualized pulmonary lesions characterized by blotchy, increased densities partially obliterating the heart shadow. Group 1 also showed blotchy areas of increased density indicating pneumonia or pneumoconiosis lesions (385, 386).

Tissues obtained from a dog that received 33 months of daily exposure to radon daughters with uranium ore dust plus cigarette smoke have been examined. All tissues except the lungs appeared normal. The lungs were extensively involved with a variety of chronic effects with a densely consolidated area consisting of a foreign body granulomatous process. The response showed accumulation of hyalin-proteinaceous appearing material which was frequently within macrophages, macrophage proliferation, giant cell formation and fibrosis and chronic inflammatory cell infiltration. Moderately severe emphysema was present. These changes were probably the result of daily inhalation of radon daughters with uranium ore dust and cigarette smoking. An exact counterpart of the granulomatous lesions has not been described in other species and therefore is not identified as a common feature of radiation pneumonitis or pneumoconiosis.

In other experiments using plutonium oxide inhalation in one dog (causing long-term alpha radiation of the lung), the presence of C-20 arachidic acid was reported and the dog had a suspect tumour. This fatty acid has also been reported in lung washings from humans with pulmonary adenomas (385, 386).

Clearance of radon daughters has been limited to consideration of clearance due to mucous movement. Such a choice assumes that the radon daughters undergo radioactive decay and deliver their dose before a significant clearance by other mechanisms has occurred. The mean clearance values are usually considered to be 1.25 cm/minute for the trachea and main bronchi; 0.25 cm/minute for the lobar (secondary) bronchioles; and 0.05 cm/minute for the segmental tertiary bronchioles. Some investigators have used a reference mucous velocity in the trachea of 1.5 cm/minute which leads to a transit time for the trachea of 8 minutes. Estimates of transit times in other regions are based on this reference value for the trachea and an assumed daily mucous production of 10 ml/day. Estimated transit times for different sections of the respiratory tract are presented in Table 140 (387).

TABLE 140

Transit Times in Different Sections of the Respiratory Tract

(according to Haque & Collinson and Altshuler, Nelson & Kuschner)

	Transit Time (min.)	
Section	**Haque and Collinson**	**Altschuler et al**
Trachea	9.6	8
Main bronchi	3.8	6
Lobar bronchi	3.04 - 7.6	11
Segmental bronchi	18 - 25.4	37
Subsegmental or intrasegmental	Long	82
Terminal	Long	1,980

(Source: Reference 387)

Most investigators appear to have used mean clearance times of 2 - 4 hours with various fractions of deposited material being removed (387).

If indeed inhaled cigarette smoke slows or stops ciliary movement, transit times would be lengthened and radioactivity would remain in contact with the epithelium longer. Recurrent tracheobronchitis which denudes patches of the epithelium of cilia would ensure that particulates bearing radon daughters would remain in contact with the bare epithelium for longer periods. In miners who have developed chronic bronchitis because of moderate to heavy smoking habits, with characteristic excessive production of mucous, could possibly find some protection from radon daughters because the mucous blanket would be so thick (thicker than 25 u) that radioactivity would not penetrate to the epithelium.

The origin of lung cancer is most likely the basal cells of the bronchial epithelium in the tracheobronchial tree. Because of the thickness of the basal cells and the mucous blanket, it is difficult to estimate the dose received by the basal cells due to the deposition of radon daughters on the mucous blanket. Some basal cells will not be irradiated at all because they will be protected by a sufficient thickness of mucous which exceeds the range of the alpha particles; others will be reached by the RaC' alpha and not the RaA alpha, while others will be within reach of both alpha particles.

One author estimated the dose to basal cells to be 24 rads per year for mouth breathing and 13 rads per year for nose breathing. Other investigators estimated the dose at the same location to be about 4 times higher (see Table 141 for a summary of exposure) (387).

TABLE 141

Summary of Published Dose Calculations for Radon and Radon Daughters[a]

Investigator	Isotope contributing to dose	Critical tissue	Calculated dose, rad/WLM
Evan and Goodman (50)	Radon only	Lung	0.002
Lorenz (51)	Radon only	Lung	0.002
Mitchell (52)	Radon only	Bronchi	0.01
Shapiro (53)	Radon plus daughters	Tertiary bronchioles	0.6
Chamberlain and Dyson (54)	Radon plus RaA only		0.4
Holaday et al (36)	Radon plus daughters	Main bronchi, bronchial tissue	2.3
Thomas (55)	RaA only	Bronchial tissue	<0.1
Altshuler, Nelson and Kuschner (46)	Daughters	Segmented bronchi	3.0
Jacobi	Daughters	Secondary-quaternary bronchioles	2.9
Haque and Collinson (48)	Daughters	Segmented bronchi	12.0
Harley and Pasternak (49)	Daughters	Segmented bronchi	0.2 - 0.3
Walsh (33)	Daughters	Bronchial epithelium	<1.0

(*) Daughters imply short-lived daughters only RaA, RaB, RaC and RaC'

(Source: Reference 387)

The identification of the most frequent site of tumour origin and the characterization of the degree of attachment and size distribution of the vector aerosol for radon daughters is crucial to the evaluation of the problem of cancer. Hyperplasia or metaplasia in the human respiratory tract may cause thinning of the bronchial epithelium, which would expose increased numbers of basal cells to the interior environment of the respiratory passages. These changes, plus the inhibition of ciliary action during cigarette smoking, may increase the absorbed dose of alpha radiation from deposited radon daughters (386).

Except for the trachea and main bronchi, over 67% of the basal cells are within range of the RaC' alpha particles. Also, RaC' alpha's contribute about 95% of the dose to the bronchial epithelium for the "typical" uranium mine atmosphere. The measurements are for normal bronchial epithelium and these properties could change by inhalation of various agents, or by the presence of pathological conditions such as chronic bronchitis, so that with changes in the lung defences these percentages could change (387). Sixty-eight percent of miners in multiple studies done with uranium miners on the Colorado plateau smoked one pack of cigarettes or more per day. Most of these miners suffered from chronic bronchitis and an increased incidence of respiratory diseases. Uranium miners have a pulmonary disease incidence five times greater than the expected rate (388). Under these conditions, clearance of particulates and radon daughters would not proceed according to the normal pattern discussed above.

If there is no clearance, most of the radiation dose will be due to the RaC' alpha. This is an important consideration since the relative concentrations of RaA, RaB and RaC in a mine are sensitive to the degree of ventilation; if the relative concentration is important, the working level exposure concept would not be valid. It is important to realize that the fraction of uncombined radon daughters is dependent upon the degree of ventilation in a mine. The reduction in dose due to a decrease in radon daughter concentration by infusing an outside air supply or by air turbulence may be nullified in part by an attendant increase in the fraction of uncombined radon daughers (RaA, RaB and RaC) as a reduction in the concentrations of radon daughters may also tend to decrease the number of available condensation nuclei (387).

A comparison of the properties of several reference atmospheres can be found in Table 142 (387).

For practical purposes, the use of the concept of the working level (WL) has been adopted by most countries around the world. It is defined as any combination of radon daughters in one litre of air which will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy. This is equivalent to the alpha energy released by the total decay of the short-lived radon daughters and radioactive equilibrium with 100 pCi of radon-222 per litre of air. One working level month is equivalent to exposure to 1 WL for one month of 170 working hours.

The external gamma radiation intensity to which miners are exposed seldom reaches 2.5 mrem per hour. The International Commission on Radiological Protection for Occupational Exposure suggests a maximum permissible dose of 5 rem per year for the whole body. Such a dose might be received by a miner exposed to 2.5 mrem per hour for over a year, but this is unlikely. Radiation film badges worn by miners enable gamma radiation exposure to be measured (383).

A considerable body of knowledge is available which supports a cause and effect relationship between exposure to radon and radon daughters, and an increased risk of developing lung cancer. Most of the knowledge that is presently available has been obtained from animal studies. From studies of mice, rats and dogs, tumours have been observed to be epidermoid carcinomas, adenocarcinomas and hemangioendotheliomas which are usually associated with localized areas of plutonium-induced fibrosis, mainly in the periphery of the lungs. Pulmonary tumours have been found in 4% of rats depositing 0.004 to 0.008 uCi of 239-Pu, whereas 20 -30% of the animals showed tumours at inhaled doses of 0.040 to 0.080 uCi. Relevant animal data is summarized in Figure 126.

In considering experimental dose-effect relationships, it must be kept in mind that lung dose estimates are usually stated as cumulative doses from the time of exposure to death. The dose required to initiate uncontrolled malignant growth is less than this by an unknown amount.

Although macrophages tend to accumulate polonium particles and thus concentrate the radiation dose, the number of cells in the lung irradiated by a given mass of plutonium dioxide particles will tend to be greater when

TABLE 142: Comparison of the Properties of Several Reference Atmosphers

Reference	Fraction of free ions			Activity on particles <0.1 um	Diffusion constant		Types of atmosphere	Particle density, cc^{-1}
	RaA	RaB	RaC		Ions	Nuclei		
Altshuler et al (46)	0.09	0.01	0.001	60	0.054	16×10^{-6}	Mine	
Jacobi (47)	0.25	0.01	0.002	50		3×10^{-5}	Normal Room Air	10^4
Haque and Collinson (48)	0.35	0.06	0.08	75	0.054	5.4×10^{-6}	Room air	3×10^4
Walsh (33)				100		13×10^{-6}	Mine	$10^4 - 10^5$
Harley and Pasternak (49)	0.04	0.002		20	0.054	1.2×10^{-6}	Mine	10^4

(Source: Reference 387)

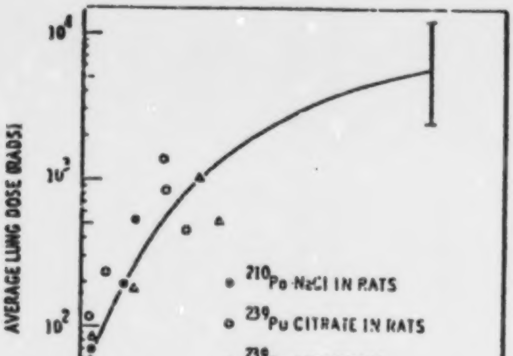

FIGURE 126

Relationship of the average lung dose to the incidence of pulmonary neoplasms in animals following inhalation of alpha emitting radionuclides. The data on ^{210}Po in rats if from Yuile et al; ^{239}Pu citrate and ammonium pentacarbonate in rats, from Antonchenko et al, and Erokhin et al; and ^{239}PuO$_2$ in dogs, from Park et al. The incidence of pulmonary neoplasms in non-exposed control animals was less than 1% in all groups.

(Source: Reference 383)

plutonium particles are smaller than when the plutonium consists of relatively large particles.

Many of the ultrastructural alterations in the lungs of rats who have inhaled plutonium dioxide particles are similar to those described following external irradiation of the lung. The alveolar alterations following ^{239}PuO$_2$ inhalation are progressively more severe with increasing lung doses ranging from 300 - 13,000 rads. Plutonium interferes with the normal cellular processes in the lung, among which the most important are those associated with alveolar macrophages. Inhaled plutonium also exhibits a cytocidal action in macrophages (389).

In one study, rats were administered $^{239}PuO_2$ particles on a single occasion and then examined throughout their lifespans. The mean aerodynamic diameter of the PuO_2 was 2.5 um. About 50% of the initial alveolar deposition of Pu was cleared during the first 30 days after exposure; thereafter, the alveolar retention half-time was about 250 days. A significant increase in malignant tumours was found only in the lung. Most lung tumours were adenocarcinomas. Squamous cell carcinomas were found, but in lesser abundance. The incidence of lung tumours was 0% in unexposed controls, 0% in the initial alveolar deposition of 0.18 nCi of ^{239}Pu, 10% in 5 nCi, 37% in 45 nCi and 53% at 180 nCi. An analysis of radiation dose to the lung indicates that a significantly increased incidence of lung tumours occurs at lung doses of greater than 78 rad (Table 143) (390).

Dust-free daughters, inhaled by animals, are mostly retained in the upper respiratory system and as a result, do not reach the lung. Thus the contribution to the lung dose is considered to come from the dust-attached daughters and radon by inhalation. The lung dose is almost entirely from alpha rays from radon (Rn), RaA, and RaC', and negligibly from beta and gamma rays (391).

In another study, ^{210}Po was adsorbed onto ferric oxide carrier particles with a mean diameter of less than 0.7 u and suspended in saline. The suspension was instilled into the trachea of hamsters at a dose equivalent to 3 mg of ferric oxide. The major radiation dose following this method of administration goes to the bronchiolar-alveolar region of the lung, the region where the majority of induced lung tumours arise. Most of the tumours were found in the peripheral lung. It was noted, that in some animals, particularly from the low exposure group in which the tumour induction times were very long, there were focal areas of marked bronchiolar epithelial hyperplasia with cellular atypia and metaplasia. These were considered to be borderline malignancies. Neither control animals nor those receiving carrier ferric oxide only developed lung cancers (392).

In a study of uranium miners on the Colorado plateau, the expected and observed bronchogenic carcinomas in a group of miners from July 1950 through December 1970 were examined. In miners exposed to 119 WLM,

TABLE 143

Incidence of Lung Tumours as Related to Radiation Dose Delivered from Inhaled $^{222}PuO_2$

		Incidence of pulmonary tumours (%)			
Cumulative dose[a] to lung (rad)	Number of rats	Total	Adeno-carcinoma	Squamous cell carcinoma	Hemangio-sarcoma
0	48	0	0	0	0
<10	131	1.5	0.8	0.8	0
27 ± 12	51	7.8	3.9	0	3.9[c]
78 ± 17	26	34.6[b]	30.8	3.8	0
255 ± 132	38	44.7[b]	36.8	5.3	2.6
680 ± 120	16	31.3[b]	18.8	6.3	6.3
2100 ± 1210	18	66.7[b]	33.3	22.2	11.1
>10,000	15	46.7[b]	6.7	33.3	6.7

[a] At 620 days after exposure; mean ± standard deviation

[b] Includes one fibrosarcoma

[c] Significantly greater than control at $P < 0.05$ level using Kolmogorov-Smirnov two-sample test

(Source: Reference 390)

there was no difference in expected versus observed pulmonary cancers. However, at a radiation level of 120 and over, there was a significant increase in the observed bronchogenic carcinomas in miners compared to the expected number.

Epidermoid, small cell undifferentiated, and adenomatous types of bronchogenic carcinoma were all shown to be increased among uranium miners. The predominance of the small cell undifferentiated histologic type was so great that it appeared to be the only type whose frequency was elevated in earlier relative distribution studies.

The same three histologic types of tumours have been shown to be elevated among cigarette smokers, but with different relative frequencies (392).

Among the criteria established to determine whether the lung cancer of uranium miners was a result of the radiation exposure in the mine, the following guidelines are used. The undifferentiated small-cell type of cancer is the predominant type that occurs among uranium miners. This contrasts with the finding that for the general population, the predominant cell type is epidermoid. Most lung cancer in miners attributed to overexposure to radon daughter products appeared between about 7 and 50 years after start of exposure. Since lung cancer appears within these time limits, this is circumstantial evidence that it is a result of the occupation. Lung cancer in uranium miners occurs with greater frequency as their mine exposure increases. It is less apparent that the greater the radiation exposure, the greater the liability to develop cancer from occupational exposure (393).

Excessive respiratory cancer has been demonstrated among all groups of uranium miners who have had more than 120 working level months of radon daughter exposure. Lung cancer incidence rises with increasing exposure. Factors which might distort this dose-response relationship are many. Exposure to other agents, such as cigarettes, probably contributed to the excess respiratory cancer. These factors should be considered in setting permissible levels. Respiratory cancers are continuing to appear at a high rate among the study group even though radon daughter levels have been markedly reduced and most of the study group has stopped mining (394).

A concern over the increased cancer rates for uranium miners has led to a new standard for the annual permissible exposure of 4 cumulative working level months (WLM). Epidemiological studies showed that lung cancer occurs at progressively lower exposure to mine environments and after increasingly longer latent periods. The authors found that there were additional cases of lung cancer among miners who had received less than 120 WLM of radon daughter exposure in uranium mines, but with 7 - 30 years of prior hard rock mining.

This finding complicates the dose-response relationship at lower exposure levels. Contaminants such as ore dust, diesel engine exhaust or blasting gases may contribute to respiratory tract pathology. In hard rock mines and uranium mines, there are often clouds of silica-bearing dusts. The diesel engines used in these mines lead to a repeated daily exposure of the miners to NO_2, SO_2 and PNA's. A report in 1969 indicated a four-fold increase of lung cancer risk for smoking miners versus smoking non-miners, who corresponded to non-smoking groups. In 1969, another report came out that the incidence of lung cancer among miners is 6 times greater than that for non-miners; the total number of lung cancer cases was 62 out of a population base of 3,414 miners (395).

Until 1970, no one realized that there was radioactivity in the Kiruna iron mines in Sweden. In most places, there were concentrations of radon daughters of between 10 - 30 pCi/l, but in some unventilated galleries, even higher concentrations were found. This was attributed to the fact that the radon had not had time to evaporate in the running water.

In general, the levels of radon measured at the time of publication were considered to be representative of earlier conditions (396).

A statistically significant excess of lung cancer cases was observed among the underground Kiruna mine employees in comparison to other workers. Since lung cancer is in excess in this relative evaluation, all other types of cancer should be low. However, the GI cancer incidence in underground miners was not low, which raised the suspicion that there may also have been an increase in mortality from GI cancers (396).

Data permitting the comparison of lung cancer frequency and exposure to daughter products of radon-222 were obtained by epidemiological studies of uranium miners performed in the United States and in Czechoslovakia (397). There are further findings in the Czechoslovakian study.

The greater part of a group of 3,366 workers evaluated, started uranium ore mining between January, 1948 and December, 1952, and the remaining between January, 1953 and December, 1957. There were three sub-groups according to age at the start of the exposure: up to 29 years, 45% of the miners; 30 to 39 years, 28%; and above 40 years, 27% of the miners.

Table 144 shows the expected, observed, and additional frequencies of lung cancer per 1,000 persons. Statistically significant differences between the observed and expected values of lung cancer were found among uranium miners at each cumulative radiation exposure category down to and including 100 - 149 WLM. Table 145 shows the dependence of the additional cancer frequency on the exposure in the three sub-groups of miners divided according to age at the start of exposure. Each of the observed values was

TABLE 144: Lung Cancer Among Czechoslovak Uranium Miners in Relation to Different Levels of Cumulative Radon Daughter Exposure -- The Total Group up to 31 December 1973

Cumulative Radon Daughter Exposure		Frequency of Lung Cancer per 1,000 Miners			
Categories (WLM)	Mean exposure with standard deviation (WLM)	Expected	Observed	Additional (0 - x) and (95% confidence limits)	Observed/Expected and (95% confidence limits)
Less than 50	39 ± 1.2	16.6	33.2	(1.8 - 16.6* - (61.0)	(1.1) - 2.0 - (4.6)
50 - 99	80 ± 0.8	13.2	21.2	(2.7) - 8.0* - (20.0)	(1.2) - 1.6 - (2.5)
100 - 149	124 ± 0.8	13.8	34.0	(9.3) - 20.2 - (31.1)	(1.7) - 2.5 - (3.3)
150 - 199	174 ± 1.2	15.2	69.8	(35.1) - 54.5 - (73.9)	(3.3) - 4.5 - (5.7)
200 - 299	242 ± 1.7	15.7	76.3	(42.5) - 60.6 - (78.7)	(3.8) - 4.9 - (6.0)
300 - 399	343 ± 3.2	17.4	102.3	(54.9) - 84.9 - (114.9)	(4.2) - 5.9 - (7.6)
400 - 599	488 ± 4.9	16.5	117.9	(70.2) - 101.4 - (132.0)	(5.4) - 7.2 - (9.0)
600 and over	716 ± 9.0	17.2	138.9	(76.2) - 121.7 - (167.2)	(5.4) - 8.1 - (10.6)
TOTAL		15.1	65.6	(43.1) - 50.5 - (57.9)	(3.8) - 4.3 - (4.8)

(*) Non-significant at 5% level

(Source: Reference 397)

TABLE 145: Absolute Risk per 1,000 Miners in Relation to Exposure Categories in Three Sub-Groups According to Age at the Start of Exposure (Group A of Miners). In Parenthesis - 95% Confidence Limits

Categories (WLM)	Additional Lung Cancer Per 1,000 Miners Per 1 WLM		
	Up to 29 years	30 - 39 years	Above 40 years
Less than 100	(0.00) - 0.12 - (0.96)	(0.01) - 0.10 - (1.64)	(0.34) - 0.58 - (2.93)
100 - 149	(0.01) - 0.10 - (0.43)	(0.08 - 0.18 - (0.77)	(0.43) - 0.54 - (1.61)
150 - 199	(0.03) - 0.11 - (0.39)	(0.10 - 0.22 - (0.69)	(0.30) - 0.72 - (1.14)*
200 - 299	(0.04) - 0.13 - (0.22)	(0.08) - 0.19 - (0.41)	(0.34) - 0.59 - (0.84)*
300 - 399	(0.06) - 0.17 - (0.28)	(0.07) - 0.25 - (0.43)	(0.13) - 0.34 - (0.55)
400 - 599	(0.07) - 0.14 - (0.21)	(0.15) - 0.29 - (0.43)	(0.11) - 0.25 - (0.39)
600 and over	(0.06) - 0.14 - (0.22)	(0.09) - 0.22 - (0.35)	(0.03) - 0.16 - (0.29)
TOTAL	(0.10) - 0.14 - (0.18)	(0.16) - 0.23 - (0.30)	(0.28) - 0.37 - (0.46)*

(*) Stat. significant difference between the younger and older age groups (P 0.05)

(Source: Reference 397)

significantly different from the expected ones at the 5% level above 100 WLM in the older age sub-group, and above 150 WLM in the middle and younger sub-groups. Significantly higher additional cancer frequency and absolute risk in the lower exposure categories were found in the older age sub-groups than in the younger sub-group. No significant differences among the three age sub-groups above 300 WLM of cumulative exposure were found. The presumption of linearity between excess of lung cancer and cumulative exposure only in the younger and middle age groups was not in contradiction with the results obtained.

In this study it was concluded that the findings supported a casual relationship between cumulative airborne radiation exposure and the excess of lung cancers among uranium miners, the principal features being an increasing ratio of observed to expected lung cancer frequency over 100 WLM in the total group and above 150 WLM in the younger groups of miners (397).

By means of repeated investigation in a random group of 700 miners, the percentage of cigarette smokers among uranium miners was found to be the same as in the general male population of Czechoslovakia. There was no reason to suppose that consumption of cigarettes correlated with the radiation exposure otherwise than to age. The use of age standardized general vital statistics permits exclusion of smoking as the major causal agent in the induction of the excess lung cancer found. The possible contributive role of smoking by affecting the distribution of radon daughters or acting as co-carcinogens cannot, however, be excluded (397).

Ionizing radiation is also regarded as not having a threshold when one considers its genetic or somatic mutagenic effects (388). Reducing the exposure statistically reduces the chance of an untoward event, such as altering genes or inducing malignant change, but it never eliminates the possibility entirely. In this situation, one attempts to get exposure low enough that untoward effects are inapparent because they happen so rarely or are too long delayed.

Hence, it is very likely that in miners the pattern is non-threshold. Evans has called it a "practical threshold". This would be a range below which most subjects would have succumbed to old age and its accompanying ills before cancer appeared. There is no doubt that a number of processes occur which undoubtedly have different response curves. The dosage to target cells would be strongly influenced by factors that interfere with the cleaning mechanism of the respiratory tract, leading to local "hot spots", or to more general slowing or overloading, which might insult the lower bronchi. Products of cigarette smoke could be important here.

One of the problems is that a dose of 1000 working level months over ten years from ages 20 - 30 would be more dangerous than 1000 working level months spread over 10 years from 50 - 60, because the miner would be around longer to develop cancer (388).

Figure 127 illustrates the dose-effect curve established by Wagner and his associates for the mortality from lung cancer in different groups and the relative cumulative dose of radiation in the workers. The curves practically coincide when standardized according to age and also to smoking and characterize the sharply-pronounced linear dependence between the annual frequency of cases of cancer of respiratory organs for 10,000 workers and the relative cumulative dose (384).

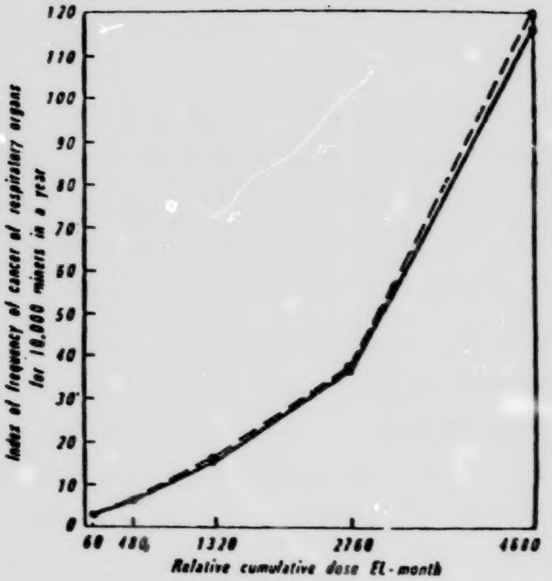

FIGURE 127

Frequency of diseases owing to cancer of respiratory organs, standardized according to age and smoking factor as a function of the relative cumulative dose -- index of frequency, standardized according to age -- according to age and smoking factor:

1-Index of frequency of cancer to respiratory organs for 10,000 miners in a year;
2 - Relative cumulative dose, EL. mouth

(Source: Reference 384)

Besides lung cancer, the influence of daughter products of radon on the respiratory organs of miners may lead to the development of fibrous processes and may aggravate pneumoconiosis, which is caused by mine dust. Archer and his associates found that with an increase in the radiation index characterizing the level of influence of daughter products or radon, i.e. the relative cumulative dose, there was an increase in the frequency of pulmonary fibrosis, accompanied with emphysema, dyspnea and constant cough. Although the important factor in the development of pneumoconiosis is related to mine dust, additional radiation of the respiratory organs of miners, especially if it exceeds the dose of 1000 - 1500 rem, may also play an unfavourable role and may be the cause of development of a mixed process -- the so-called silico-radiation pneumosclerosis (384).

One study was conducted in Ontario investigating "Radiogenic Lung Cancer in Ontario Uranium Miners 1955 - 1974". Eighty-one deceased miners formerly on the Ontario Nominal Roll died of lung cancer during 1955 to 1974 and were compared with 81 surviving miners from the Nominal Roll. It was noted that the deceased cancer victims had undergone exposure to an average 74.5 WLM while the survivors were exposed to an average of 32.8 WLM. It was also determined that there was a dependence of lung cancer risk on dose, existing within the range of doses experienced by the described uranium miners (Table 146).

The ratio of cancer cases to survivors increased as cumulative exposure increased, suggesting an increase in lung cancer risk with increasing dose (398)

Radon daughter levels present in Ontario uranium mines are uncertain in the period prior to March 1957 when regulations went into effect.

Twenty-eight miners from the Nominal Roll died from lung cancer in the period 1958 to 1974 who had commenced uranium mining in 1958. For this group the cumulative exposure varied from 0 to 225 WLM, with the mean being 31.1 WLM. This compares favourably with the surviving miners' mean of 32.8 WLM. Table 147 was drawn up to determine if a detectable dependence of lung cancer risk on dose exists for this sub-group of lung cancer victims.

TABLE 146

Radiogenic Lung Cancer in Ontario Uranium Miners 1955 - 1974

Cumulative Exposure	No. of Cancer Cases	No. of Survivors
0 - 29 WLM	30	48
30 - 89 WLM	26	24
90 + WLM	25	9

(Source: Reference 398)

TABLE 147

Radiogenic Lung Cancer in Ontario Uranium Miners 1958 - 1974

Cumulative Exposure	No. of Cancer Cases	No. of Survivors
0 - 29 WLM	18	16.6
30 - 89 WLM	8	8.3
90 + WLM	2	3.1

(Source: Reference 398)

Based on the mean cumulative exposure and the number of cancer cases for different cumulative exposures, there appears to be no discernible difference between the post-1957 cancer case group and the original survivor group (398).

The overall mortality experience suggests that cancer of the lung fatalities are 1.8 times the expected rate (81 actual, 45 expected). Comparison of the 81 cancer victims with the survivor group in the Royal Commission report indicates that the risk of dying of cancer by 1974 is 2.5 times as great for persons entering the Ontario uranium mines before 1958, relative to those who entered after 1958. These facts, when associated with the comparison of cancer victims who started work after 1957 and the survivor group, suggest that the post-1957 and pre-1958 exposure groups were significantly different. It is the authors' contention that this difference is due to the under-estimation of existing radiation levels in Ontario uranium mines in the period preceeding 1958 (398).

It is predicted of the survivor group that 46 of 81 (56.8%) Ontario uranium miners began mining during or after 1958. The workers entering the mines after 1957 are assumed to have the same risk of dying from lung cancer in the period 1957 -1974 (in the absence of any occupational exposure to radiation) as the entire group; the expected number of lung cancer cases in the post-1957 group can be calculated from the overall expected lung cancer deaths and the survivor group percentage. Using this assumption, 25.6 deaths from lung cancer would be expected in the study period. The Nominal Roll shows that 28 miners commencing work after 1957 died of lung cancer in the study period. In the post-1957 group, actual deaths due to lung cancer exceeded the expected by less than 10% (398).

The analysis just outlined suggests that miners entering Ontario uranium mines after 1957 have little (less than 10%) increased risk of dying from lung cancer in the study period (398).

On the basis of the above statistics and the moderately limited time span of twenty years, if current Ontario ventilation standards are followed, radon-induced cancer of the lungs may no longer be an important and fearsome concern in the uranium mines of Ontario. However, this pro tem conclusion can only become firm if further study is applied over the next decade until valid statistics are available for the full thirty-year latency period associated with the development of lung cancer in the uranium miner.

Until such time as a firm conclusion can be derived, action should be taken to prevent smokers from working in uranium mines and to lower the level of diesel particulate below 2 mg/m^3, preferably to 1.5 mg/m^3, or less.

The French Atomic Energy Commission now has available a personal radiation dosimeter for use with uranium workers (399). Using the recommendations of the ICRP, the instrument allows the measurement of radon daughters, thoron daughters, long-lived dust and gamma rays. The personal dosimeters continuously sample the ambient atmosphere. Alpha rays are emitted by short-lived radon and thoron daughters and collected on filters in the dosimeter. After waiting a suitable period for the short-lived elements to decay, the filter is placed into an instrument and counted for long-lived dust. Also, a TLD chip can be inserted into the head to measure gamma exposure.

Until recently, radon exposure data has been exclusively obtained by dosimetry of the collective type. Consequently, exposures were calculated from radon concentration data or alpha energy levels collected by grab sampling in the working area. The accuracy of these calculations has been queried and it is difficult to accurately predict levels in a particular working area. The observation of workers and inspectors over the years led to the conclusion that exposure was probably underestimated when using collective dosimetry. Therefore, the development of personal or individual dosimeters was considered important.

A number of studies were conducted from 1978 to 1981 in five underground mines and the conclusion was reached that individual dosimeters were a more adequate form of equipment for measurement. Monthly measurements of miners were obtained simultaneously with individual alpha dosimeters with collective dosimetry being done as a comparison. An underestimation factor was defined as the ratio of the collective exposures in five mines, expressed as man X joules, obtained by each type of dosimeter. An example of comparative exposure values is given in Table 148. The last column gives the underestimation factor for collective dosimetry, which was generally in the range of 1.5 - 2.0-fold.

TABLE 148

Mine Identification Number	Number of Exposures Obtained by Both Dosimetries	Underestimation Factor	Grab Sampling Based On
A	1760	1.4	222 Rn
B	90	1.9	222 Rn
C	40	1.8	222 Rn
D	40	1.9	222 Rn
E	40	2	E_α

(Source: Reference 399)

4.13 Cigarette Smoking

Cigarette smoking accounts for a large excess of mortality and morbidity due to lung cancer and chronic airway obstruction, as well as cardiovascular and peripheral vascular disease. Cigarette smoke contains a complex mixture of particulates and volatile substances, several of which have biologic actions. For example, "tar" contains several chemical substances which can induce cancerous lesions experimentally when painted on the skin of a rodent. Nicotine in smoke may be responsible for pharmacologic dependence to cigarettes; other constituents of smoke release histamine from lung tissue, stop the beating of cilia, delay particle clearance from the lungs, cause reflex bronchoconstriction, induce changes in alveolar macrophage cells and their function, and cause cytotoxic effects in lung explants.

Long-term smokers often have chronic cough, sputum production and increased dyspnea on exertion, as well as a greatly increased risk of lung cancer. Function studies show airway obstruction, loss of diffusing capacity, and decreased arterial oxygen. After only 3 to 5 years of smoking, teenagers have an excess of respiratory symptoms and decreased flow rates on maximum expiratory flow volume curves.

The pathology underlying the functional changes in young cigarette smokers is probably a respiratory bronchiolitis, which may be a precursor of centriacinar emphysema. Progression of airway obstruction in people who continue to smoke is inferred from the severe disease found in older smokers.

In the United States, coronary heart disease is the leading cause of death and the largest contributor to excess deaths among cigarette smokers. Cigarette smoking independently of, and synergistically with, other coronary heart disease factors greatly increases the risk of developing coronary heart disease (400).

When mortality from all types of death were determined for 1974, those for cigarette smokers were 64% higher than for non-smokers; in contrast, the overall age-related mortalities for cigar and pipe smokers were only 16 and 11% higher, respectively. The statistical data available related to the excess cancer death caused by cigarette smoking in the United States in 1974 is shown in Table 149. The excess deaths were calculated by the following equation:

$$E = \frac{D.f(m-1)}{1+f(m-1)}$$

where E = excess deaths due to cigarette smoking, f = fraction of smokers in the population, m = mortality ratio of smokers to non-smokers for a specified type of cancer, and D = the number of deaths from that type of cancer.

This equation considers the number of smokers who would have developed cancer of the respective site in the absence of smoking. The value of f is an average value over the years relevant to the final deaths, typified by the figures for 1965 when 52% of males and 34% of females smoked. Some degree of uncertainty with regard to the exact value of the mortality ratio, m, must remain unresolved. When this ratio is high, as in the case of bronchogenic cancer, the difference in the number of deaths attributed to smoking varies surprisingly little with variation in m (400).

In Hammond and Horn's study (401), the death rates over 44 months from specific causes were studied in relation to smoking habits in a group of 187,783 men between the ages of 50 and 69. During the period of the study, 11,870 of these men died. For microscopically-proven cases of cancer, as well as for the total cases reported as cancer, it was found that the death rates were higher among regular cigarette smokers than among men who never smoked, that the mortality ratio increased with the number of cigarettes smoked per day, and that the death rates were higher among pipe and cigar smokers than among men who never smoked. A total of 7,316 deaths occurred among regular cigarette smokers, an excess of 2,665 over 4,651 that would have occurred had the age-specific death rates for smokers been equal to that for non-smokers.

Coronary disease accounted for 52.1% of the excess; lung cancer accounted for 13.5% and cancer of other sites for an additional 13.5%. An extremely high association between cigarette smoking and death rates for men with lung cancer was found in rural areas as well as in large cities. The most important finding of this study was the high degree of association between cigarette smoking and the total death rate (401).

Anderson and Ferris (402) surveyed 1,261 residents of a town by respiratory questionnaires and forced expiratory volume (FEV_1) tests.

TABLE 149: Excess Cancer Deaths Caused by Cigarette Smoking In the United States in 1974

Type of Cancer	Deaths in 1974 D	Fraction of smokers f	Mortality ratio m of smokers to non-smokers	Excess deaths due to smoking E	(%)
Bronchogenic[a]	78,873	0.43[b]	11	63,966	81.1
Laryngeal	3,262		5.5	2,150	65.9
Oral[c]	7,968		4	4,494	56.4
Esophageal	6,652		4.5	3,998	60.1
Bladder	9,397		2	2,819	30.0
Pancreatic	17,376		2	5,213	30.0
Renal	7,073		2	2,122	30.0
TOTAL	130,601			84,762	64.9
All types of neoplasms[d]	360,472			84,762	23.5

[a] Includes carcinoma of trachea, bronchus and lung.

[b] Smoking in U.S. adults in 1965; Past history influences cancer incidence.

[c] Includes malignant neoplasma of buccal cavity and pharynx.

[d] Includes benign neoplasms and lymphatic and hematopoietic malignant neoplasms.

(Source: Reference 400)

Men smoking more than one package of cigarettes a day had a lower FEV_1 at each age group than non-smokers or those smoking less. The men who smoked one package or less a day had a mean FEV_1 similar to that of non-smokers until the age of 54 and similar to smokers of more than one package a day after that. There was a consistent increase in all chronic respiratory disease, chronic bronchitis and irreversible obstructive lung disease with increasing current and lifetime cigarette exposure. After age standardization, the cigarette smoking effects remained highly significant, where the effect of age after standardization for smoking was much less (402).

Pipe and cigar smokers were found to have a greater risk of disease than subjects who had never smoked tobacco. A male-female ratio for all chronic respiratory disease was 1.85. The ratio may be the result of greater male occupational exposure to noxious dust and gases in the population surveys. However, the prevalence of irreversible obstructive lung disease was the same in both sexes for the subjects smoking more than one package a day over the same lifetime cigarette smoking exposure.

It is apparent that cigarette smoking is clearly associated with the prevalence of chronic respiratory disease. Because of this, standardization for smoking must be made in any epidemiologic study. Non-smokers may be more suitable subjects for studying the effects of atmospheric pollutants or occupational exposure to noxious dusts or gases (402).

The effects of numerous environmental factors, physiological or atmospheric, on tracheal mucous drainage, both in vivo and in vitro have been studied (403). This involves the precise measurement of the rate of ciliary beating in beats per minute or cycles per minute.

As inspired air temperature increased, the frequency of ciliary beating increased. When the pH increased into the alkaline levels, the ciliary beat also increased. Acid pH is inhibitory to ciliary movement, whereas an alkaline pH up to 10 exerts an activating effect on the cilia.

Ciliary activity remained unaffected by variations of partial pressure of oxygen from 7 to 3000 torr, but ciliary activity is significantly reduced when the partial pressure of carbon dioxide exceeds 35 torr.

The cilia are extremely sensitive to irritant pollutants, in particular sulphur dioxide, nitrogen oxide, chlorine, short-chain aldehydes, and cyanohydric acid. A number of these substances are present in high concentrations in the gaseous phase of cigarette smoke. Ciliary activity is markedly slowed down, with resulting stagnation of the mucous blanket of the airways, in direct proportion to the length of time of exposure to these pollutants. It is extremely interesting to note that 4 hours of exposure to ozone in two concentrations failed to change the ciliary activity (403).

The reaction of cilia to the pollutants may explain, in part, the toxic action of cigarette smoking on mucociliary function. The particulate phase of cigarette smoke contains pollutants that inhibit ciliary function and mucous function. Cigarette smoking, by reducing the rate and function of the mucociliary action, enables infectious and cancerogenic agents to attack the mucosa (403).

To examine the transport mechanism in smokers, Camner and Philipson (404) used a test aerosol with particles of fluorinated ethyl propylene in the form of an aqueous dispersion to determine tracheobronchial clearance. Six smokers were studied who smoked 15 to 20 cigarettes a day, and none of them had experienced any irritation in the respiratory tract prior to the testing. No clear tendency was found to reduce the clearance in connection with acute exposure to tobacco smoke. There was, however, a consistently slower clearance about a week after the subjects stopped smoking.

These results are in contrast to animal studies in which slower clearance was observed in connection with acute exposure to tobacco smoke. It is probable that exposure in the animal studies was much greater. A reasonable working hypothesis would be that tobacco smoke first stimulates mucociliary transport, only to inhibit it if the dose increases beyond a certain limit (404).

By 1978, many studies had been done attempting to demonstrate that cigarette smoke acts as a carcinogen in humans. Supporting studies with animals have been criticized for unrealistic exposures of cigarette smoke given to animals. For example, a common "carcinogen" in cigarette smoke is benzo(a)pyrene and this has, on occasion, been given in doses amounting to many milligrams. Benzo(a)pyrene values have been reported in the range of 0.3 to 12.25 ug per 100 cigarettes.

However, human epidemiological studies have shown that there is not only a dose-response relationship between cigarette smoke and lung cancer, but the induction period can be shown to be shortened as the daily doses of cigarette smoke increase with time. Furthermore, there is a downward shift in age of diagnosis if the age at the beginning of smoking decreases. It has been calculated that a person who smokes one package of cigarettes per day for 30 years may be exposed to a total dosage of 0.66 to 26.83 mg of benzo(a)pyrene in the maintstream smoke. Investigators in this field of research feel the evidence is very strong that smoking is one of the causes of lung cancer (405).

It has been established that alpha-emitting radon daughters increase the risk of bronchial cancer in underground miners. Naturally-occurring polonium-210 and its parent, lead-210, are found in tobacco smoke and increased polonium activity is found in bronchial tissues of smokers versus non-smokers. It has been postulated, therefore, that polonium-alpha radiation may be a significant cancer initiator in cigarette-induced bronchial cancer (406).

In examining the overall increase of cancer of the respiratory tract which has developed since the 1950's, the factors most implicated are cigarette smoking, urban pollution and the emergence of new industrial environments. The common element to these three factors are the polynuclear aromatic hydrocarbons (PAH), which have demonstrable carcinogenic potency in animal studies.

As methodology is rapidly becoming available, widespread studies are beginning to provide meaningful data. Studies in the United States as well as in Czechoslovakia report high PAH pollution in smoke-filled rooms. In some African tribal huts where fires are burned continuously and the interior is always smoke-filled, there is a significant increase in nasal and lung cancer.

As more sophisticated analytical methods for the assay of carcinogens become available, it may become apparent that the majority of the carcinogens in polluted air and cigarette smoke are PAH's and the majority of these are derived from the incomplete combustion of organic matter.

Hoffmann and Wynder (407) found that the burning of materials rich in aromatic hydrocarbons leads to relatively high yields of carcinogenic hydrocarbons and volatile phenols. The aromatic portion of gasoline is claimed to be the precursor of the carcinogens emitted into the environment and it is suggested that the organic content of gasoline be decreased in order to minimize the PAH emissions. Combustion products of plants, especially those of leaves such as tobacco, have been identified as providing carcinogenic hydrocarbons including alkylated fluorenes, fluoranthenes and carbazoles.

With regard to tobacco carcinogenesis, the majority of carcinogens in tobacco smoke have been shown to be on the particulate matter. Large scale fractionations and bioassays of "tars" have demonstrated that most carcinogens and tumour accelerators are concentrated in the neutral fraction, which amounts to about 0.6% of the dry particulate matter.

The importance of PNA's in cigarette smoke as tumour initiators is clearly indicated by correlations between the tumour yield on mouse skin and the BaP concentration in the tar of commercial and experimental cigarettes (Figure 128). Benzo(a)pyrene serves only as an indicator for the concentration of the pyrosynthesized carcinogenic hydrocarbons. For nitrate-rich natural tobaccos or tobaccos enriched with KNO_3, a correlation was found between the concentration of nitroalkanes, nitrobenzenes and PNA in the smoke and the nitrate content of the tobacco. This supports the concept that PNA's in combustion products are formed by way of C-H radicals. Thermally-activated nitrogen oxides are formed from alkali nitrates in the burning zone, react as scavengers for C-H radicals, and form nitrohydrocarbons. The concentration of C-H radicals is reduced by this competitive reaction (407).

It has been well documented that in order for BaP to be maximally carcinogenic, it must be metabolized by non-specific cytochrome P-450 mediated aryl hydrocarbon hydroxylase (AHH) to reactive intermediates, which can then bind covalently to nucleic acids and proteins of various tissues. Lubawy and Perrier (408) investigated the effect of whole smoke or the gas phase from smoke on influencing the metabolism of BaP by an isolated perfused rabbit lung. In untreated rabbit lungs, radioactive BaP disappears from plasma perfusing the isolated lungs according to first-order

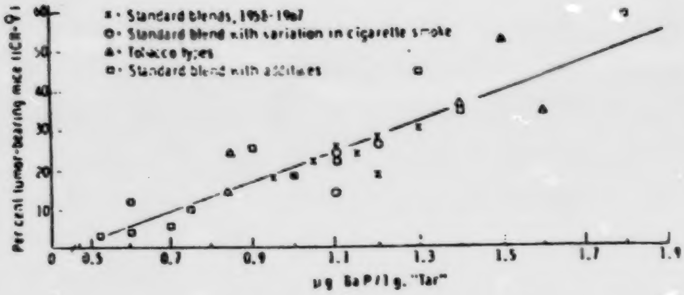

FIGURE 128

Bioassay of cigarette smoke condensates tumorigenicity and BaP

(Source: Reference 407)

kinetics. However, when these lungs are ventilated with freshly generated whole cigarette smoke, the disappearance curves are altered dramatically. It was also shown that the gas phase from smoke had little effect, indicating that it was the particulate phase of cigarette smoke which was critical to the disappearance of the BaP. The results suggested that the acute effect of whole smoke on BaP metabolism is inhibition mainly due to the presence of particulate matter. The relevance of this work to the metabolism of BaP by smokers' lungs is not clear.

Beta-naphthylamines, a proven carcinogen to man, is present in cigarette smoke and this ingredient should be considered in relation to tobacco carcinogenicity. The mainstream smoke of one 85 mm non-filter U.S. commercial cigarette was found to contain 27 ng alpha-naphthylamine and 22 ng beta-naphthylamine.

Furthermore, it should be recalled that a large number of N-nitrosamines are highly carcinogenic to experimental animals and often only a small dose of nitrosamine is needed to induce tumours in animals. For example, 15 ug of diethylnitrosamine has produced tumours in the respiratory tract of hamsters. Tobacco smoke has been shown to contain trace amounts of nitrosamines in nanogram quantities.

As a result of these findings, it is apparent that there are multiple carcinogens in cigarette smoke (407).

In a 1975 summary of the findings of a recent Third World Conference on Smoking and Health, Steinfeld (409) reported that cigarette smoking is considered the number one cause of lung cancer in industrialized countries. For all smokers, the risk of developing lung cancer compared with non-smokers ranged from 7.6 to 14.2 times as great, and for men who smoke heavily, the risk of developing lung cancer was 4.9 to 23.9 times that of non-smokers. Cigarettes exert an effect on the incidence of coronary heart disease and hasten the onset of angina. It is suggested components of cigarette smoke may play a synergistic role in the development of coronary heart disease.

Moreover, chronic bronchitis and emphysema, which are both included in the term chronic obstructive pulmonary disease, are about 18 times more common among smokers as compared with non-smokers. Mortality rates from these diseases are 4 to 25 times greater in smokers than in non-smokers (409).

Wanner and his colleagues (410) exposed purebred adult beagle dogs to tobacco smoke for 13.5 months (equivalent to 100 cigarettes per week). The smoke was administered through the mouth and nose for 1.5 hours twice daily and was inhaled every tenth breath in a manner to simulate human smoking. Twenty-four hours after the last exposure, the tracheal mucous velocity (TMV) was measured. Four animals served as controls and were observed for 4 months. A significant decrease in TMV occurred in dogs exposed to cigarette smoke, compared with the control animals. Lung compliance and resistance, single-breath diffusing capacity, lung volumes, and arterial PO_2, PCO_2 and pH did not differ significantly between the two groups. The authors concluded that suppression of mucociliary activity associated with cigarette smoking may precede other abnormalities of pulmonary function (410).

Syrian hamsters were exposed twice daily, 5 days a week to 8 puffs of cigarette smoke with a concentration of 19.3% smoke that lasted 15 seconds a puff with 43 seconds between each puff. In exposed hamsters, macrophage granulomas were seen in the pulmonary parenchyma. Occasional malignant tumours were found in the nasopharynx of smoke-exposed animals.

Dysplastic changes were seen in the larynx after approximately 40 weeks of smoke exposure. Neoplastic changes were also found in the larynx of smoke-exposed animals, beginning at about 80 weeks of exposure (411).

The effect of extracts of cigarette smoke or cigarette tobacco, on the in vitro oxygen consumption of pulmonary macrophages, was studied by York and his co-workers (412). Smoke extract caused a progressive inhibition of respiration as a function of concentration. Tobacco extract, on the other hand, produced a biphasic effect, with stimulation at low concentrations and inhibition at higher concentrations. Cell viability was affected by both types of extracts.

Cigarette smoke exposure depressed clearance of particles and bacteria presumably as a result of specific effects on intracellular biochemical mechanisms operative in macrophages. Secondly, pulmonary macrophages possess lysosomal proteolytic enzyme systems which were presumably not only important for intracellular digestion of engulfed materials, but were also hazardous to lung tissue. Therefore, under certain circumstances, macrophages may release their lysosomal enzymes extracellularly and provoke tissue damage to both pulmonary, cellular and interstitial tissue constituents (412).

The effect of acute, intensive cigarette smoking on respiratory function was investigated by McCarthy et al (413) in subjects who had smoked 28 ± 10 cigarettes per day for 12 ± 9 years. Abstention from smoking occurred for 1 to 24 hours before the study commenced. The results showed a significant decrease in FVC, FEV_1 and PEF, but there was no significant change in midmaximum expiratory flow rate (MMEF) or MEF at vital capacity 25% (VC_{25}). The changes in lung function that occurred after acute smoking did not appear to be related to age or to the status of pulmonary function before the study. No correlation was observed between the degree of change of lung function tests and the smoking history of the subjects to the number of cigarettes smoked during the acute session. The data indicates that acute intensive smoking is associated with a reduction in so-called effort-dependent tests, as well as in alterations in gas mixing in the lung. There may be some variation due to effort or fatigue, but this would not account for the changes in peak flow or FEV_1.

This study was unable to confirm significant changes in other tests that reflect alterations in the small airways, which is considered typical of the earliest and most significant changes seen in disturbed lung function of smokers. The failure to see changes in MMEF and MEF at VC_{25} would indicate that either the changes are not related to changes in the small airways or the tests undertaken were not significantly sensitive to indicate the changes that actually occurred. It should be noted that the MMEF and MEF at VC_{25} did not improve in the group of smokers that were restudied 6 months after modification of smoking habits (413).

An investigation was undertaken by Jorgensen in Sweden of workers in a mine where diesel-driven vehicles were used (414). Half of the subjects were underground workers, while the control group consisted of surface workers. Half of each group were smokers and half were non-smokers. A younger group, with an average age of 30 years, was compared with an older group with an average age of 55 years.

In the underground group, more workers, predominantly the smokers and the older workers, reported that the air was "bad" compared to the surface group.

The frequency of bronchitis was higher among underground workers than surface workers, much higher among smokers than non-smokers, and also higher among older than among younger workers. The effects of underground work and smoking seemed to be synergistic.

Respiratory failure studies did not reveal any difference between the groups and sub-groups other than that which could be accounted for by differences in age (414).

In 1953, a major epidemiological investigation was carried out in the British coal fields (415). The purpose of the study was to determined whether a relationship existed between dust exposure and occupational pulmonary disease. The survey was still ongoing in 1975. The relationship between pneumoconiosis and the FEV_1, vital capacity and forced expiratory volume as a percentage of vital capacity were examined separately; all 3 was related to radiological abnormality. Cumulative exposure to coal mine dust in the respirable range were calculated from dust sampling results over a 10-year period, together with estimates of the previous exposure. The index of pulmonary ventilation was the FEV_1.

The conclusions were that there was a progressive reduction in FEV_1 with increasing cumulative exposure to airborne dust. Among men with simple pneumoconiosis, there was no evidence of a reduction in FEV_1 in excess of that attributable to their dust exposure, smoking habits, age and physique. Simple pneumoconiosis appeared to be evidence of the amount of previous dust exposure and suggested that this had been sufficient to cause a measurable fall in forced expiratory volume. The extent of ventilatory impairment associated with simple pneumoconiosis was less than that due to age alone in most men. Cigarette smoking was found to be an important cause of ventilatory impairment (415).

Battigelli et al (416) in 1964, investigated a group of locomotive repairmen exposed to diesel exhaust and compared them to a control group of railroad workers matched for age, body size, etc., but having the least occupational contact with diesel exhaust. No difference between exposed and control groups was found with respect to frequency of complaints and with regard to chest abnormalities. The same results were observed for various pulmonary function tests carried out on the two groups of workers.

Concentration of diesel exhaust constituents showed NO_2 levels of about 0.5 ppm during the winter and about half that amount in the summer. Median sulphur dioxide levels were just under 1 ppm, and total hydrocarbons were 2 ppm. The average period of exposure of the diesel workers was 9.6 years.

In contrast to the negative conclusions bearing on the relationship of diesel exhaust to pulmonary function impairment, the comparison of the entire group divided into smokers and non-smokers revealed marked differences. The majority of the various complaints reported for the whole group (both exposed and control) came from smokers. This was evident in reference to cough, sputum and a combination of complaints. There was a consistent finding of better pulmonary test performance in non-smokers (416).

The effect of smoking cessation or modification on lung function was determined during a 48-week period (417). In a large number of the cigarette smokers, a disturbance of lung function was not revealed by conventional tests, such as the FEV_1; however, an abnormality was often demonstrable in tests that may reflect alterations in small airways such as Phase III of the single-breath expired N_2 curve. There was no relation between the degree of abnormality of lung function and the presence of respiratory symptoms, but there appeared to be a relationship to lifetime smoking history, the heaviest smokers having the poorest function.

Cessation or more than a 25% decrease in the number of cigarettes smoked was attended by improvement in the slope of Phase III of the N_2 washout curve, as well as FVC and FEV_1. Resumption of smoking after cessation was attended by a deterioration in the slope of Phase III. The data suggest that the functional abnormalities in smokers are related to smoking and are at least partially reversible by cessation or significant modification of the smoking habit (417).

A study was undertaken by Buist et al (418) to obtain more information about the effect on lung function of stopping smoking or of modifying the smoking habit, and to determine the time course of change. A group of 75 cigarette smokers who attended a smoking cessation clinic were studied using a respiratory symptom questionnaire and spirometry. Subjects were tested before stopping smoking and at 1, 3, 6 and 12 months after the initial testing. There was a dramatic decrease in respiratory symptoms in those who stopped smoking, and a moderate decrease in those who reduced their consumption by at least 25% (418).

The literature relating to the health effects and increased mortality due to various causally or possibly causally-related diseases and cigarette smoking presents considerable argument in favour of the value of cessation of smoking. Statistical and epidemiological data support the fact that if a patient has smoked more than 20 cigarettes a day for 25 - 35 years, his risk of death can still be materially reduced by cessation of smoking, and very significant health gains are apparent if he quits smoking at age 45 - 54 years. In addition, increased reduction of disease-specific mortality for several of the specific entities associated with cigarette smoking can be made following cessation of cigarette smoking (419).

Cross-sectional studies suggest that termination of cigarette smoking by individuals results in improvement of pulmonary function and diminution or erradication of chronic respiratory symptoms. Several studies showed that smoking subjects who abstained for 6 weeks, had increased peak flow rates, diffusing capacity, inspiratory reserve volume and maximal voluntary ventilation. Heart rate, oxygen debt after exercise and functional residual capacity were decreased (419).

Studies by Wagoner, Saccomano, Archer and Holoday demonstrated that in the uranium mines of the Colorado plateau, smoking uranium miners had eight times the likelihood of developing lung cancer when compared to non-smoking uranium miners (420, 421). Uranium miners are most likely to succumb to undifferentiated small cell carcinoma, the most virulent of the lung cancers, and carrying the worst prognosis (422, 423, 424). This type of tumour is one of the most frequently found cancers in the general population of smokers (425, 426, 427).

Fifty to sixty percent of miners are moderate to heavy smokers, with the inevitable respiratory impairment of chronic bronchitis, recurrent respiratory insults of viral bronchitis and bacterial infections after three to eight years of smoking (428, 429). By going underground to conditions where even the most stringent ventilation and decontaminating regulations are put into effect, the intercurrent levels of pollutants such as SO_2, particulates, PAH's and radon daughters can adversely affect the smoking miner's impaired airways and incompetent immunological defences so that he faces increased respiratory morbidity as well as increasing chances of earlier cardio-respiratory mortality.

It should be noted that mining is, like many occupations, somewaht selective in that those with the least respiratory problems, and those that react less seriously to the effects of cigarette smoking are the miners who stay on for decades at underground work.

Nevertheless, the escalating respiratory morbidity and mortality suggest that the greatest hazard a miner faces is his own habit of cigarette smoking. It would seem that the toll of human disease and mortality could be reduced if regulations prevented current smokers from working underground.

4.14 Epidemiology

Epidemiological studies are probably the most informative way to approach the acquisition of data on the long-term health effects of air pollution, be it ambient air to the general population or in working environments such as in underground mines. However, as noted by Hiller and Bone (482) the interpretation of the data is made difficult because of subtle physiological changes which may result from other etiologies, such as smoking or allergies, and which could lead to the same physiologic changes in individuals.

It is in the epidemiology of diesel exhaust component health effects where the greatest deficit of knowledge exists. As will be clear from the summary of the available data presented in this section, there are no definitive studies available which provide 'hard' scientific information to definitely delineate diesel exhaust as being carcinogenic or even in causing a significant reduction in pulmonary function.

The highest risk of lung cancer is found among men age 45 - 70 years who are heavy cigarette smokers exposed to certain occupational carcinogens. A 20-year study in Great Britain has provided accurate estimates of the risk. This group was not exposed to any extent to occupational risk factors. The risk of cancer amongst smokers compared to non-smokers was 10-fold, with there being a 14-fold greater risk among cigarette smokers and a 25-fold greater risk amongst those smoking 25 or more cigarettes a day. Most of the risk of lung cancer accumulates after the age of 45 (430).

In a random sample of the Canadian population aged 45 - 64 years approximately 6 cases of lung cancer per thousand men would be expected and just over 1 case per 1000 in women, without regard to smoking status. In 1975 the Department of National Health and Welfare indicated that 46.5% of Canadian men and 31.8% of Canadian women aged 45 to 64 years were regular cigarette smokers. Therefore, 6.9% of men and 2.1% of women who smoke 25 or more cigarettes a day could expect 53 cases of lung cancer per 1000 men and 21 cases per 1000 women over the next 5 years.

A large group of occupational agents have been identified as increasing the risk of lung cancer in Canada, such as the exposure to asbestos in insulation workers, miners, millers, textile workers, shipyard workers, construction workers and demolition crews. Polycyclic aromatic hydrocarbons are believed to be responsible for the increased risk in a number of other workers. Radioisotopes, particularly radon daughters, are also recognized as causing lung cancer in uranium miners. Studies by Selikoff and Hammond have served to draw notice to multiple risk factors of lung cancer both in Canada and Britain. Selikoff and Hammond found, using a questionnaire, that from 1967 -1974 there was a relation between smoking habits and death from lung cancer. There was apparently no increase in risk for workers who had never smoked cigarettes or had smoked only pipes or cigars, but the risk was nearly 6 times for those who smoked cigarettes, as well as for those whose smoking habits were unknown. This last group was believed to consist mostly of cigarette smokers. Using data from Finland, Higginson found similar ratios of 20:1 which confirmed a multiplicative relationship between tobacco smoking and asbestos exposure. This multiplicative relation can be expected from the less well-documented experience in uranium miners. Therefore, if any preventive measures are going to be taken it should be aimed at men aged 45 - 64 years who smoke 25 or more cigarettes a day and are exposed to occupational agents such as radiation, asbestos or nickel (430).

Since the greatest risk for lung cancer is in men aged 45 years or more who smoke at least 25 cigarettes a day and who were exposed for at least 20 years to particular occupational agents such as asbestos, radiation or polycyclic aromatic hydrocarbons, a workshop held in March 1979 considered ways of preventing lung cancer in these individuals. Two alternative approaches were considered for the prevention of lung cancer and these included immunoprophylaxis and the use of retinoic acid derivatives (431).

Immunoprophylaxis has been used in preventing other disease entities, including the use of other vaccines for rabies and whooping cough. There is little evidence that immunotherapy is effective for viral or bacterial diseases. Immunotherapy is only effective if the tumour burden is minimal and the animals immune status is healthy. Some immunotherapy has been instituted for stage 1 lung cancer but has not proven effective in humans and its usefulness is yet to be assessed.

However, for immunoprophylaxis of adults at high risk the proposal is to inject tumour-associated antigens. Such antigens are immunogenic, that is they stimulate the immune defense system of humans, and would be derived from pools of cancers such as squamous cell cancer, adenocancer and anaplastic large and small cell carcinomas.

The side effects would include some small ulcers which might be unacceptable to healthy men (431).

One question raised was whether or not intradermal immunization of healthy adults, without lung cancer, would provide the cellular surveillance that would effectively recognize a small tumour cell population with minor antigen changes when such a tumour cell population arose.

It was agreed that in adults of a very high risk population, who have already shown marked cellular atypia in their sputum, the problem is really one of diagnosis and treatment rather than prophylaxis (431).

Different problems arise with the use of retinoic acid derivatives in the prevention of lung cancer in high risk adults. It was felt that since the use of retinoic acid is a short-term treatment, the hope of a long-term preventative effect would not be realized, and consequently this product would have to be taken indefinitely. When a pharmacological dose is used for acne, some toxic effects are seen and this might be a limiting factor. However, what was not discussed was whether or not less than the pharmacological doses used in acne would be appropriate for the prevention of lung cancer. There was some doubt expressed by the investigators as to the most suitable tumour to study in animals to better understand the prevention attained by the use of such agents.

Generally it was agreed that further work was needed and that a pilot study should be developed for the specific study of these particular problems (431).

It is interesting to note that Goldsmith (432), in discussing epidemiology and environmental health policies, emphasized the high frequency of lung cancer in cigarette-smoking uranium and asbestos workers and suggested the need for policies which would exclude cigarette smokers from entrance into these occupations, a recommendation put forth relative to mine workers in dieselized underground mines by the present authors.

Waller (433) presented preliminary results of a long-term study of the incidence of lung cancer among London Transport workers, including mechanics, etc., who worked in bus garages with high concentrations of diesel fumes. Although the absence of data on smoking habits made it difficult to determine whether the small differences in rates between job categories were significant, the standardized mortality ratios were well within the range of those found in National studies of lung cancer mortality and smoking in relation to occupation. As well, Waller pointed out that although there has been an increased use of diesel equipment in the London area and thus a supposed increase in diesel pollutant levels in the atmosphere of the city, the lung cancer mortality in England and Wales has been declining over the last few years. These data are not definitive but are suggestive that diesel pollution does not lead to an increased incidence of lung cancer.

Calabrese and colleagues (434) evaluated the 9 major epidemiological studies that have been completed where an attempt was made to determine the influence of occupational diesel exhaust on human populations.

The studies of Raffle (435), Kaplan (436) and Waxweiler (437) were reviewed in relation to lung cancer, morbidity and mortality. Unfortunately, the limitation on these earlier studies is that widespread dieselization of transportation and mining operations did not generally occur until the 1950's or later. Thus, exposure periods were generally for less than 25 years, which may not be sufficiently long enough to demonstrate any increase in lung cancer mortality. More extended latency periods are usually needed for such an evaluation.

The Raffle (435) study found no excess lung cancer in diesel-exposed bus mechanics between 1950 - 1954. However, the study had many methodological limitations. No control for the smoking habits of workers was included, and diesel exposure levels were not well documented. Nontheless, there was no marked change in the annual rate of lung cancer in the workers.

Caplan's study also found no association between diesel exposure and lung cancer in railroad workers. Again, there was no control for smoking habits of workers and the individual exposure to diesel exhaust was not well documented.

The Waxwhiler et al study (437) found no significant mortality differences between diesel-exposed and non-exposed potash miners. The causes of death considered included a number related to the respiratory tract, as well as malignant neoplasms. The maximum diesel exposure in this study was only 17 and 10 years, for the exposed and non-exposed groups, respectively.

There has been only limited research to assess the relationship of diesel exhaust to specific morbidity due to respiratory involvement, such as bronchitis, asthma, influenza and infection. These diseases are prevalent in the general population and are of special significance for the very young and the elderly.

El Batawi and Nowier (438) and Jorgensen and Svensson (439) investigated respiratory morbidity. El Batawi and Nowier found a higher frequency of upper respiratory tract diseases, peptic ulcers, gastritis and high blood pressure in diesel bus mechanics in Alexandria, Egypt than in other occupational groups. Again, there were methodology deficiencies such as no comparison or control group, an extremely high proportion of heavy smokers and night shift workers which makes it difficult to assess the effects of diesel exposure.

Jorgenson and Svensson (439) investigated respiratory morbidity in diesel exposed iron miners. The data appeared to indicate that there was a synergistic effect between smoking and exposure to diesel mine environments. However, the exposure of diesel exhaust cannot be separated from the effect of exposure to the mine environment as a group of surface mine workers was used as a control.

According to Calabrese et al (434) a large body of literature suggests that there is an association between diesel exposure and reduced pulmonary function and increased non-specific pulmonary complaints. Reger and Hancock (440), in a study of over 700 diesel-exposed coal miners, matched with non-diesel exposed coal miner controls, found the diesel-exposed miners had an increased presentation of cough, mucous, and exacerbation of previous respiratory symptoms. The exposed workers also had slightly poorer pulmonary function. The disparity of symptoms of pulmonary function between exposed and non-exposed workers was found to increase with the length of employment. However, decreases in pulmonary function did not consistently relate to the extent of diesel exposure.

Interestingly, wheezing and shortness of breath were reported more frequently amongst control miners. This study was limited by the innate problem of diesel exposure being compounded by exposure to the mining environment, both for the control and for the diesel-exposed miners. As well, the controls were selected from a group of mine-exposed workers 10 years prior to the exposed workers. This may not be a major problem, but possible changes in smoking habits, levels of tar in cigarettes and other variables were not taken into account in the study.

Reger, Hankinson and Merchant (441) examined 60 diesel-exposed workers from a matched pair study, in which workers were from a broad spectrum of mining jobs. Although there was an absolute decrease for 5 pulmonary function parameters in the exposed miners compared to non-exposed, none of the between-group differences were found to be significant. Population matching was imperfect and resulted in an older control group with a longer mean underground exposure.

Gamble et al (442) tried to examine the relationship between exposure to NO_2 and changes in pulmonary function over the course of a work shift in five U.S. salt mines. Levels of NO_2 were found to be directly related to changes in pulmonary function for four of five pulmonary function tests considered. The Gamble et al study suggested that decriments in pulmonary function may also be early indicators of increased susceptibility for irreversible lung disease.

Battigelli et al (443) found no significant differences in pulmonary function or respiratory complaints between diesel-exposed railway house workers and non-exposed yard workers. In this study, smoking was not well controlled for and the control population was found to have a higher smoking index than the exposed group.

Calabrese et al concluded, from their review of the literature, that:

(1) there was no evidence of excess cancer mortality due to exposure of diesel fuel exhaust. However, the studies considered have to be interpreted with caution because in each study the methodology had definite limitations,

(2) there is insufficient evidence to evaluate the relationship between exposure to diesel exhaust and other lung disorders, including chronic bronchitis and respiratory infection, and

(3) some evidence suggests that exposure to diesel exhaust could be associated with reduced pulmonary function. However, the long-term significance of this reduction has not yet been evaluated.

Attfield, Trabant and Wheeler (444) completed an epidemiological study of 6 potash mines in New Mexico in 1976. The mines used diesel equipment to one extent or another. However, only Mine C appeared to have a significant number of diesel-powered units of greater than 50 HP. The participating miners at each location were administered a questionnaire on respiratory symptoms, work history and smoke habits. As well, each underwent a posterior-anterior and lateral chest radiograph and had simple spirometry completed for determination of maximum values of FVC and FEV_1. General air quality in the mines was evaluated with total dust exposure through personal gravimetric samplers, as well as diesel soot, hydrocarbon, temperature and humidity measurements being taken. Because Mine C was the mine with the most extensive use of diesel exhaust, most of the comparisons in the report were with Mine C compared to Mine E, which had the least diesel equipment. The total dust concentration in Mine C was 23 mg/m^3 compared to 18 mg/m^3 in Mine E, but interestingly, as a result of the increased diesel use, the NO_2 concentration in Mine C was 3.3 ppm compared to 0.1 ppm in Mine E. The ratio of respirable to total dust in Mine C was 3, compared to 11 in Mine E. Carbon monoxide levels in Mines C and E were 9 and 5 ppm, respectively. Overall, no statistically significant differences between the workers from either of the mines could be detected for lung function in either smokers or non-smokers. There did appear to be some differences in that non-smokers in Mine C had worse than predicted FEV_1 values and both smokers and non-smokers had an increased incidence of phlegm and cough than was predicted. The authors concluded that apart from there being no true effects of potash and diesel exhaust, negative findings could have resulted because of poor data, particularly in exposure estimates, from statistical power and because of selection effects. Although the authors searched for reasons why negative data was obtained, they were unable to establish a trend between measures of diesel use, NO_2 exposure, dust exposure and worsening of health.

Ames et al (445, 446) completed respiratory evaluation of 60 diesel-exposed and 90 non-diesel exposed coal miners for pre- and post-workshift ventilatory function changes. General matching case and control subjects were obtained from the dieselized and non-dieselized mines. There was an overall statistically significant workshift-coal mine dust decline in FVC and FEV_1 for both the diesel and non-diesel exposed groups. The workshift change decreases in ventilatory function occurred particularly in cigarette smokers. The smoker-non-smoker contrast equalled or exceeded workshift-coal mine dust effect changes for the diesel exposed and non-diesel exposed workers. Furthermore, no significant relationship was found between the level of NO_2 exposure and ventilatory function changes over the workshift in either diesel-exposed or non-diesel-exposed workers.

Jones, Gamble and Minshall (447) completed an industrial hygiene-epidemiological study of workers at 4 diesel bus garages which had varying diesel exhaust levels. Some buses were "running" repair facilities and others were for major overhauls where engine running time was low and the number of engines entering and leaving the facility in any short period of time was minimal. Various diesel contaminants, including SO_2, sulphates, sulphites, several aldehydes, PNA's, phenol, hydrocarbons, CO, NO, NO_2, nitrosamines and particulate were measured in the garage atmosphere. At the same time, workers at these two facilities were administered pre- and post-shift pulmonary function tests, chest x-rays and a respiratory questionnaire. Except for NO_2 and CO, the level of contaminants, when measured individually, were below the existing OSHA or NIOSH recommendations. Levels of NO_2 and CO did reach 1 and 50 ppm, respectively, in specific instances. Interestingly, with a detection limit of about 1 ng/m^3, nitrosamines were not detectable in any samples in Garage D, which included a running repair area. PNA's which were sampled for (limits of detections in brackets), included BaP (0.0061 - 0.4 mcg/m^3), crysene (0.015 to 0.13 mcg/m^3), pyrene (0.015 to 0.26 mcg/m^3), B(a)A (0.0061 to 0.066 mcg/m^3), fluoranthene (0.015 to 0.052 mcg/m^3) and cyclohexane solubles (30 mcg/m^3). Benzo(a)pyrene was detected in only 3 samples collected, in a mechanics bay and two garages, but the levels were just at or only slightly above the limits of detection. All other samples were below the limit of detection. The current OSHA standard for CO is 50 ppm (TWA exposure). In two of the garages, the TWA averages were about 25 and 40 ppm. Therefore, it is at least possible that some mechanics were exposed to CO levels higher than the NIOSH recommendation of 35 ppm. Similar trends were seen for the NO_2 levels. One garage had the highest constant level with a weighted average of about 1 ppm. In a running repair area, NO_2 remained fairly low except for occasional peaks to about 2 ppm, which coincided with buses being started in the vicinity of the meter. The current OSHA standard for NO_2 is 5 ppm, but NIOSH recommends that levels should be maintained below 1 ppm. The area sampling in the garages would suggest that the concentrations of NO_2 were well within the OSHA requirements, but would have exceeded the NIOSH recommendations on occasion. SO_2 and NO_2 adsorption to particulate was examined and the data suggested that these gases did not tend to be associated with diesel particulate under the conditions of study. The aerodynamic mass median diameter of the particulate present in the garages ranged from 0.7 to 3 um, with an average geometric standard deviation of about 6. The range of particulate size appeared quite high, and these data seem to conflict with other reports indicating that most diesel particulate is below 0.5 um in diameter. Two reasons were given for this discrepancy, the first being that samples were not pure diesel particulate since they were collected in general areas throughout the garages. Road dust could have been integrated along with the diesel particulate, which would tend to give it a higher median diameter. Secondly, it was apparent that the diesel aerosol tended to agglomerate which would result in higher mean diameter values.

In a companion report, Gamble, Jones and Minshall (448) reported the epidemiological portion of their study on the acute effects of diesel exhaust on the respiratory system of workers in the diesel bus garages. Two hundred and thirty-two workers in the four garages were examined before and after a work shift. Pulmonary function was assessed by determining if the mean changes were significantly different from 0, and secondly, dose-response relationships were analyzed by multiple regression techniques. One-half of the study population were smokers, 22% were ex-smokers, and 28% were non-smokers. Eye irritation was the most common symptom reported and showed the strongest association with the exposure variables. Well over half the study subjects complained of eye irritation related to work, but there was no apparent decriment in pulmonary function associated with this symptom. The prevalence of cough (46%) and chest tightness (13%) were not elevated compared to lead battery workers given the same type of questionnaire. Other symptoms, such as headaches (29%), wheeze (15%) and difficult or laboured breathing (14%) were greater compared to the lead battery workers. There was no association of acute reductions in pulmonary function with personal measures of NO_2 or respirable particulate. Although smokers consistently had the largest reduction in pulmonary function over a shift, the changes were not significantly greater than that of ex-smokers and non-smokers. The results of the study suggested that NO_2 and the particulate phases of diesel exhaust were below the threshold for producing an acute reduction in lung function. There were no statistically significant reductions in FVC, FEV_1, peak flow or flow at 50 and 75% of FVC, nor was there any detectable association of exposure to NO_2 or respirable particulate with acute reductions in pulmonary function.

Schenker (449) recently discussed the question of the potential carcinogenicity of diesel exhaust and concluded that the epidemiologic evidence did not help resolve the question of the clinical significance of diesel exhaust constituents in the workplace or environment. He suggested that the experimental evidence for the carcinogenicity of diesel engine exhaust was strong, but definitive epidemiologic studies were needed to relate the experimental evidence to humans.

In 1983, Environmental Health Associates Incorporated of California, completed a study on cancer and mortality among members of a heavy construction equipment operators union with potential exposure to diesel exhaust emissions. The study was carried out under the sponsorship of the Co-ordinating Research Council (483, 484).

The mortality study (483) was carried out on a cohort of 34,156 male members of a heavy construction equipment operators union who were members for at least one year between January 1964 and December 1978. The vital status as of the end of December, 1978 was determined for 95% of these union members of which 85% were still living, and 10% were deceased. Death certificates were obtained for 97% of all those who died. Although historical environmental measurements were not available, partial work histories were reviewed for some cohort members. The comparison employed the standardized mortality ratio (SMR). Overall mortality for the entire cohort was significantly lower than expected, with an SMR of 81.4. Out of the 3,345 deaths observed, 817 were a result of malignant neoplasms, which is slightly less than the expected value based on U.S. white male cancer mortality rates with an SMR of 93. Site-specific cancer mortality rates indicated 23 deaths were due to cancer of the liver, when only 13.8 were expected (SMR of 166.7). Three hundred and twenty individuals died from cancer of the respiratory system which calculated an SMR of 96.9, slightly less than predicted. Mortality from emphysema for the entire cohort was significantly elevated with an SMR of 165.3 being calculated as a result of 116 deaths when only 70 were expected.

The SMR for emphysema suggested an upward trend with increasing duration of membership. For those with less than 5 years, the SMR was 99, but increased to a high of 194.6 for those with 15 - 19 years membership and then reduced to 174.8 for the membership of 20 years or longer.

Cause-specific mortality rates were calculated for retirees, which included any member who retired during the study period. There were 6,678 retirees of which 1,345 were known to have died. The SMR for all causes of death was 114.5 which is statistically significant.

For cancer of all sites the MSR was 145.3 which is also statistically significant. When specific sites of cancer were reviewed, an SMR 142.4 was found for cancer of the digestive system, 181.8 for cancer of the large intestine, 162.4 for cancer of the respiratory system, 164.1 for cancer of the lung and 231.2 for lymphosarcoma and reticulosarcoma. There was also a significant mortality excess due to non-malignant respiratory disease, primarily emphysema.

The overall mortality of the heavy construction men was considered not to be unusual, as it represented the "healthy worker effect".

Although this study is of some interest, it is doubtful that the significant findings in increased emphysema and/or specific site cancer can be absolutely related to diesel exhaust exposure. Previous work histories were not available for the cohort and as well smoking and dust exposure histories could not be considered. Even though these limitations were discussed by the authors of the report, they concluded that there was "suggestive evidence for the existence of several potential health problems in this cohort". The authors suggested that a case-control investigation be completed to identify the specific work conditions or lifestyles as well as smoking history associated with those instances where there were mortality excesses.

In a companion report (484), additional elaboration of the cancer incidence from the same historical prospective study were discussed. The California Tumor Registry identified 175 primary cancer cases among union members during the study period. The most common primary sites were the digestive system (42 cases) and the respiratory system (51 cases). Using the standardized incidence ratio (SIR) where an SIR of greater than 100 indicates an excess in incidence greater than the incidence expected in the general population. For all sites combined, the SIR was 92.7, slightly less than expected, but not statistically signifciant. The SIR for the oropharynx was 711.2, but this was based on only 3 cases of cancer and 0.4 were expected. The SIR for stomach cancer was 132.4, but this was not statistically significant. The SIR for respiratory cancer was 101.3. There were non-significant differences in the SIR's for prostatic and renal kidney cancers. Therefore, except for oropharyngeal cancer of which the data were based on an exceptionally small number of cases, the study did not reveal any significant relationship between the incidence of specific sites of cancer in this group of heavy construction workers with potential exposure to diesel exhaust.

Harris (485), a statistician at MIT, recently completed a National Research Council (U.S.) evaluation of the potential risk of lung cancer from diesel engine emissions. In his statistical analysis, he reviewed the available epidemiological evidence as well as comparative analysis of laboratory and epidemiological data on diesel engine emissions and the two chemically-related environmental exposures, coke oven emissions and roofing tar emissions. Re-evaluation of the London Transport Authority data by Harris indicated that the "undetected incidence of lung cancer among diesel bus garage workers was no greater than 160% of the incidence of lung cancer among other unexposed employees" at the 95% confidence level. He also calculated that for a male aged 40 - 65, who had been exposed to an average increment of 1 mcg/m^3 of diesel particulate for 20 years, the upper confidence limit of risk was a 1% increase in lung cancer incidence. For the same group exposed to an average increment of 10 mcg/m^3 for 30 years, he calculated the upper confidence limit of risk to be a 15% increase in lung cancer incidence. By comparison, a male aged 40 - 65 who has smoked cigarettes for a comparable period had a proportional increase in the risk of lung cancer ranging from about 1,000 to 2,000 percent. This review of the available epidemiological and laboratory data is of considerable interest but still falls short of being definitive as many assumptions, some very doubtful, had to be used in the predictive calculations. Personal communication with Harris, during which he was informed that the miner working in a dieselized atmosphere could easily be exposed to 1 mg/m^3 of diesel particulate for 8 hours a day for 20 years, provided an upper limit of increased lung cancer risk of 5 - 6-fold compared to the normal population. However, given that there is considerable data to suggest that, at least in short-term bioassays of mutagenicity, particulate matter from heavy-duty diesels has a lower mutagenicity than coke oven emissions, roofing tar emissions and small diesel engines than the increased risk of 5 - 6-fold may be larger than the true value.

The Lovelace Inhalation Research Group (486) also carried out an evaluation of the potential health effect of exposure to diesel exhaust. Using a similar approach to that of Harris and combining an evaluation of both the available epidemiological and laboratory data. It must be borne in mind that all of these risk assessment articles were primarily aimed at the concerns of the potential increased use of the light-duty diesel vehicle and its effect on ambient air quality and the health of the general population, not on the use of the heavy-duty diesel engine in an enclosed underground space with limited exposure in terms of time and numbers of individuals. The publication by the Lovelace Group is an excellent review source for this important topic.

To summarize, the Group used two linear models to estimate lung cancer risk. One used the average air concentration of B(a)pyrene by people in the different groups as an index of their lung cancer risk. The second model used the average air concentration of inhaled particles as an index of risk. However, it was shown that the calculation based on exposure to BaP greatly overestimated the lung cancer risk and was for people exposed to diesel vehicle exhaust particles. They also evaluated the risk factor from the same epidemiological studies used by Harris for coke oven workers, roofers, smokers and non-smokers, etc. The reported annual cancer risks in these different groups of people is shown in Figure 129. Using this figure, if a diesel particulate exposure of 1,000 mcg/m^3 is fitted to the available data, the incidence of lung cancer in the miner is estimated to be about 110 per 100,000 workers. This compares to 7 and 3 per 100,000 individuals for urban and rural non-smokers, respectively. Smokers (10 -40 per day) have a lung cancer incidence of 50 - 80 per 100,000.

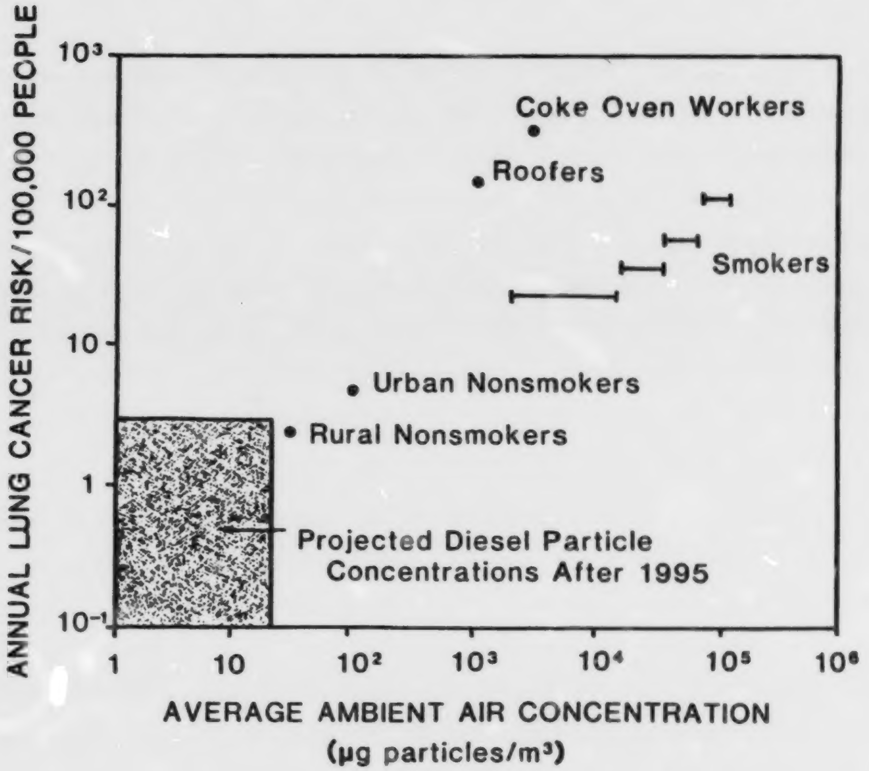

FIGURE 129

Annual lung cancer risk measured in coke oven workers, roofers, smokers and non-smokers related to the concentrations of particles inhaled averaged over all of the air breathed during their lifetimes

(Source: Reference 486)

5.0 SUMMARY AND CONCLUSIONS

In 1978, while under contract to CANMET, French & Mildon completed a review of the world literature relevant to the potential health effects of diesel exhaust on underground mine workers. They concluded that, of the vast number of pollutants which had been identified in diesel exhaust (Table 149), diesel soot or respirable combustible dust was the pollutant of greatest health concern. Diesel soot is a carbonaceous material which is virtually completely respirable and therefore is carried into the alveoli or air exchange compartments of the lungs. Thus, it was thought that the soot itself might result in physical impairment of lung function by causing fibrosis leading to a form of restrictive pulmonary disease. Of even greater concern was the propensity of soot to adsorb other chemical components such as the mutagenic/carcinogenic polynuclear aromatic hydrocarbons (PAH's), gases such as SO_2 and NO_2 and acids, including H_2SO_4 and HNO_3, and thereby act as a carrier to deliver these pollutants into the functional air exchange portions of the lungs, with the consequent development of lung pathology and/or a reduction in pulmonary function.

TABLE 149 Estimated Pollutant Levels in Dieselized Mines(1)

	Exposure Values		TLV	
	ppm	mg/m^3	ppm	mg/m^3
CO_2	400 - 1100	720 - 1980	5,000	9,000
CO	0 - 20	0 - 22	50	55
NO	1 - 5	1.2 - 6	25	30
NO_2	0.1 - 0.5	0.2 - 0.4	3	6
SO_X(2)	0.5 - 0.7	1.25	2	5
H_2SO_4	--	0.2	--	1
RCD(3)	--	2.0	--	2(4)
RDP(5)	--	0.1 - 1.0	--	1.5(6)

In addition, there are a number of other pollutants present, including benzene, aromatics, hydrocarbons, aldehydes, PNA's, organic acids, nitric acids, etc.

(1) Modified from reference NIOSH Diesel Workshop (1977), Morgantown, West Virginia.
(2) Dependent on sulphur content of diesel fuel used.
(3) Respirable Combustible Dust.
(4) No TLV available. Authors recommend 2.0 mg/m^3.
(5) Respirable Diesel Particulate.
(6) Consider RDP to be 75% of RCD.

The potential health effects of the major diesel exhaust components can be summarized, as follows:

Carbon dioxide levels in dieselized mines are directly related to the amount of diesel fuel burned. The potential for toxicity is related to a reduction in oxygen. If the oxygen level is maintained at 16 - 18 volume % even relatively high concentrations of CO_2 pose no problem. Carbon dioxide is not considered a health hazard in the mine.

Carbon monoxide reduces the O_2-carrying capacity of hemoglobin by displacing O_2 from binding sites. It has the potential to cause physiological responses in many tissues, including nerves, muscle and brain. Mine levels of CO seldom, if ever, exceed the TLV of 5 ppm and CO is not thought to pose an acute or chronic health problem in dieselized mines.

Of the oxides of nitrogen, NO_2 is more toxic than NO. The effect of NO results primarily by reducing the O_2-carrying capacity of hemoglobin, in a manner similar to CO. Underground concentrations appear to be well below the TLV of 25 ppm and thus are not a health concern.

NO_2 is an irritating gas which can cause tissue edema, tracheal constriction, reduced pulmonary function, including, with chronic exposure, emphysema, slowing of ciliary activity which can lead to impairment of the mucociliary clearance system and an emphysematous-like response. Because excursions over the present TLV of 3 ppm occur, close attention to this pollutant is required.

Chronic exposure to sulphur dioxide results in increased airway resistance (bronchoconstriction), increased mucous flow, and reduced particulate clearance from the bronchi. Small airway hyperplasia and an emphysema-like response have been observed in animals.

The adverse health effects of SO_2 are increased by the presence of atmospheric particulate matter. Up to 25% of the emitted SO_2 may be converted to sulphuric acid if a catalytic purifier is fitted; a portion of which attaches to diesel soot and is carried into the alveoli.

There is only limited data available on the effects of acid mists, but it is irritating and evidence suggests it could lead to an increase in air flow resistance and reduced mucociliary transport. It may also be partially responsible for the emphysematous-like response to SO2 exposure.

The necessity to draw from a wider variety of crude oil stocks has produced an upward trend in fuel sulphur content. Since this results in increased SO_2 emissions, levels of this contaminant are of concern. Chronic inhalation studies in dogs at relatively low concentrations of 0.5 ppm showed some decrement in pulmonary function, with development of restrictive lung disease compatible with pulmonary interstitial fibrosis.

Respirable diesel particulate (RDP) is synonomous with diesel soot. Most particles are less than 1 micron in diameter and as such can be deposited in the lower, functional exchange portions of the respiratory tract. Recent animal evidence suggests that chronic exposure to concentrations of diesel particulate in the range found in dieselized mines can cause fibrosis of the interstitial pulmonary tissue, especially at sites of macrophage clusters, resulting in a reduced lung volume and a reduction in diffusing capacity. At higher concentrations the residence time of particulates in the lung increases due to impairment of pulmonary clearance mechanisms.

With chronic exposures to rats and monkeys, doses of RDP in the range of 0.75 to 2.0 mg/m^3 have resulted in small decrements in pulmonary function and the development of interstitial disease of the restrictive type.

Of additional concern is the transport of mutagenic and carcinogenic polynuclear aromatic compounds (PNA's) into the pulmonary system adsorbed to diesel particulate, thus increasing the risk of lung cancer. The carcinogenicity of RDP has not yet been statistically demonstrated, although studies carried out by GM found tumours in 1/20, 2/20 and 1/20 animals in the 0.25, 0.75 and 1.5 mg/m^3 RDP groups compared to 0/20 in control animals.

As indicated, although CO_2, CO and NO are toxic at high concentrations, the data available on mine ventilation, diesel exhaust control systems, and mine concentrations of the substances themselves suggest that, except in rare instances, these gaseous pollutants are controlled within the current recommended ACGIH threshold limit values (TLV's). Therefore, although their presence and control are recognized, they are not considered to be the pollutants most likely to represent a health hazard to the miner. The diesel exhaust pollutants which were, and which remain, of greatest concern from a health standpoint are NO_2, SO_2 and diesel particulate. Although there was little scientific data available at the time of the original report on the toxicity of diesel particulate, the authors elected to use the conservative public health approach and considered diesel particulate to be a major health concern until such time as evidence to the contrary became available.

An equation was proposed which was variously called the Air Quality or Health Effects Index, as follows:

$$\frac{CO}{50} + \frac{NO}{25} + \frac{RCD}{2} + 1.5\left[\frac{SO_2}{3} + \frac{RCD}{2}\right] + 1.3\left[\frac{NO_2}{3} + \frac{RCD}{2}\right]$$

where the gaseous components are expressed in ppm and the respirable combustible dust (RCD), which is estimated to be approximately 75% diesel soot, is expressed in mg/m^3. It was proposed that an AQI/EQI value of 3 or less was acceptable. Values over 3 indicated an increased health risk, which required either a decrease in diesel activity or an increase in mine ventilation.

Because the formula does not truly define health effects, but is more of an estimate of the relative environmental condition in the mine as a result of diesel use, the equation, when applied to the ambient mine air, is now termed the Air Quality Index (AQI). When used to discuss undiluted exhaust, it is referred to as the Exhaust Quality Index, or EQI.

The index (Figure 130) used the ACGIH additive principle for the presence of multiple pollutants. Because of the potential synergistic effect of SO_2 and NO_2 with diesel particulate, factors of 1.5 and 1.2, respectively, were included in the equation to express these concerns. Thus, the equation is heavily weighted towards respirable combustible particulate, which appears in the equation three times. The rationale for these synergism terms are non-scientific in their origin and are "best guess" estimates to express the authors' concerns for the synergistic toxicity of particulate matter with SO_2 and NO_2. It is obvious if the respirable combustible dust in the mining environment is reduced, the AQI will be lowered substantially.

A TLV for RCD of 2 mg/m^3 was proposed, with 75% or 1.5 mg/m^3 of that being considered to be diesel particulate matter. Furthermore, a TLV of 3 ppm for SO_2 was recommended and this value has recently been reduced to 2 ppm by the ACGIH. These figures were derived from discussions held at the NIOSH Diesel Workshops held in Morgantown, Virginia in 1977 and represented the summary opinion of a number of experts. At the same

FIGURE 130

The Air Quality Index (AQI)

$$\text{AQI} = \frac{(\text{CO})}{\text{TLV-CO}} + \frac{(\text{NO})}{\text{TLV-NO}} + \frac{(\text{RCD})}{\text{TLV-RCD}} + 1.5\left[\frac{(\text{SO}_2)}{\text{TLV-SO}_2} + \frac{(\text{RCD})}{\text{TLV-RCD}}\right] + 1.2\left[\frac{(\text{NO}_2)}{\text{TLV-NO}_2} + \frac{(\text{RCD})}{\text{TLV-RCD}}\right]$$

In addition, no individual component was to exceed its TLV;

CO = 50 ppm
NO = 25 ppm
NO_2 = 3 ppm
SO_2 = 3 ppm (now 2 ppm)
RCD = 2.0 mg/m^3 determined gravimetrically, which was considered to be equivalent to 1.5 mg/m^3 R.D.P.

If the SO_2 and NO_2 concentrations are 25% or less of their TLV's, the respective ratios are not included in the calculation.

Interpretation of the AQI:

AQI = <3 : Acceptable health risk
= 3 - 4 : poses a moderate threat to health
= >4 : serious health threat requiring increased ventilation or a reduction in RCD

meeting a mining environment workshop group agreed that the pollutant levels shown in Table 149 were typical of those found in dieselized mines.

In the five years since the publication of the AQI/EQI formula, there has been a considerable body of laboratory data generated which confirms the authors' original concerns for the significant potential of diesel particulate to have deliterious health effects. A number of major research projects related to the toxicity of diesel emissions have been completed, or are nearing completion, by such groups as the General Motors Biomedical Science Department, the Lovelace Inhalation Research Institute, the Southwestern Research Institute, Battelle Laboratories, EPA, NIOSH and other research groups in the United States, Europe and Japan. The importance of diesel particulate in the view of these research groups, relative to the other components of diesel exhaust, is apparent when it is noted that in the majority of studies the concentration of diesel particulate was the major pollutant used to control exposures to the animals. Most of the studies were designed to respond to the concerns of the EPA as to the significance of the expected North American increase in the use of light-vehicle diesels on ambient air quality and the possible subsequent health effects to the general population. Comparative data on the composition of exhaust from light and heavy-duty diesel engines indicates that, qualitatively, there is little to differentiate them and thus as long as pollutant concentrations in any experimental setting are clearly defined, the toxicological data derived are relevant to heavy-duty diesel engines as well.

It is fortuitous that the GM research group conducted their diesel toxicity studies at particulate levels of 0.25, 0.75 and 1.5 mg/m^3, which are in the range considered to be typical of those measured in the dieselized mining atmosphere. Thus, their research efforts are particularly pertinent to the concerns of this report.

Since the 1978 document was distributed, several criticisms of the AQI formula were received, with one of the most prominent being that the ACGIH recommends that the additive approach for toxic compounds only be used when the components exert their toxicity by similar mechanisms. It was suggested that it was not appropriate to include the respirable combustible dust, SO_2 and NO_2 terms together with the CO, CO_2 and NO factors. A second criticism was that the synergism factors 1.5 and 1.2 for SO_2 and NO_2, respectively, were not scientifically supportable. The authors accept both these criticisms and agree that the AQI could be modified. However, as was made clear in the 1978 publication, the original AQI formula was a best estimate and it was suggested that it be put into use in a non-regulatory manner to determine the usefullness of the AQI approach. According to Mogan, Johnson and others, the AQI has utility in delineating the effect of exhaust modification systems and/or ventilation procedures in mines, by providing an indication of whether changes would lead to an atmosphere with an increased or decreased health risk (491). It also permits evaluation of the relative toxicity of the emissions of different engines which, with an economic evaluation of engine modifications, ventilation costs, etc., providing a useful way of determining the most suitable choice. As pointed out by Dainty and Mogan (491), the smaller the EQI, the less the comprehensive toxicity of the emissions and the smaller the recommended value of the ventilation when it is defined as:

Ventilation air rate = (dry exhaust gas flow rate) x EQI/3

where 3 represents the maximum desired level of the index for the working mine atmosphere (i.e.) the maximum AQI.

Interestingly, for untreated exhaust this expression yields ventilation rates similar to those recommended by the U.S. Mining Enforcement and Safety Administration (MESA) under Title 30, Part 36 of the Code of Federal Regulations. Thus, the original AQI/EQI formula does not represent a radical change in ventilation requirements from those presently required for untreated diesel exhaust.

To demonstrate the usefulness of the equation, Dainty and Mogan (491) calculated the EQI for a number of naturally aspirated, 4-stroke engines at full rated load and speed, using fuel with a sulphur content of 0.5% and a ventilation residence time of 30 minutes (to determine NO to NO_2 conversion values). Brake specific ventilation values (Table 150) permits a comparison of EQI derived values and current ventilation practice as outlined by MSHA requirements. For engines with a realtively normal emission spectrum (A, B', D, E and F) the ventilation requirement, as calculated using the EQI, does not differ dramatically from those calculated using the MSHA approach. In fact, since the emission levels quoted in the table are maxima, and thus must be multiplied by a duty cycle factor to predict mine ambient concentrations, deviations from the MSHA prescription as a working standard are even lower. With engines with a high soot output (Engines C and F'), however, the EQI approach requires that additional ventilation is required compared to the MSHA calculated ventilation values.

Having continued the review of the world literature for the past 5 years, during which time a considerable body of data relating to the carcinogenicity, mutagenicity and toxicity of diesel exhaust and diesel particulate has been generated, the authors' concerns for the importance of RCD have been reinforced. However, as indicated above, modifications to the AQI might be in order providing an acceptably useful approach to evaluating the quality of the dieselized mining atmosphere.

TABLE 150

Brake Specific Ventilation Range For Some Naturally-Aspirated Heavy Duty Diesel Engines

Engine	RCD mg/m^3	NO ppm	NO_2 ppm	SO_2 ppm	EQI 3	MSHA[1] Dil'n Ratio	Brake Spec. Vent. cfm/bhp	MSHA Brake Spec. Vent.[2] cfm/bhp
A (PC)	38	665	0	221	75	53	150	105
B (DI)	30	1490	0	213	98	119	220	265
B' (DI)	31	740	0	213	75	59	165	130
C (PC)	69	400	40	216	92	35	200	77
D (DI)	96	1041	0	191	118	83	290	205
E (PC)	31	478	0	205	65	38	150	88
F (PC)	61	658	0	209	87	53	200	120
F' (PC)	135	608	0	221	133	49	280	103

[1] MSHA dilution ratio: $(NO + NO_2)/12.5$

[2] MSHA brake specific ventilation:

$$\left[(NO + NO_2)/12.5\right] \times \text{exhaust gas volume (dry)/rated power}$$

It is now suggested that 2 equations be used, as follows:

AQI (gas): $$\frac{(CO)}{TLV\text{-}CO} + \frac{(NO)}{TLV\text{-}NO} + \frac{(NO_2)}{TLV\text{-}NO_2}$$

The AQI (gas) should not exceed 1, and no individual component should exceed its TLV.

AQI (Part.): $$\frac{(RCD)}{TLV\text{-}RCD} + \left[\frac{(SO_2)}{(TLV\text{-}SO_2} + \frac{(RCD)}{TLV\text{-}RCD}\right] + \left[\frac{(NO_2)}{TLV\text{-}NO_2} + \frac{(RCD)}{TLV\text{-}RCD}\right]$$

It is recommended that the AQI (particulate) value should not exceed 2.0, and no single component should exceed its TLV, as dictated by current ACGIH values.

In addition to "splitting up" the equation so that toxicants with similar mechanisms of action are included in individual equations, the synergism factors have been deleted because of a lack of scientific support for their inclusion. The inclusion of the RCD/SO_2 and RCD/NO_2 factors still weights the equation towards diesel particulate, which gives sufficiently significant recognition of the impact of diesel particulate and its additive or synergistic health effects with SO_2 and NO_2, to obviate the need for using additional factors.

To compare the impact of this proposed change in the AQI formula, the EQI/3 values, calculated using the 1978 equation, in Table 150 for the various engines is repeated in Table 151 alongside the EQI/2 figures calculated using the "new" AQI formula.

TABLE 151

Comparison of EQI/3 and New EQI/2 Values for Ventilation Requirements

Engine	EQI/3	New EQI/2
A	75	73
B	98	121
B'	75	68
C	92	90
D	118	116
E	65	62
F	87	87
F'	133	142

It is clear from this comparison that both approaches yield virtually the same values and thus are basically equivalent.

As a further comparison, if concentrations of pollutants for a "typical" dieselized mine atmosphere are used (i.e. CO = 10 ppm; NO = 3 ppm; NO_2 = 0.3 ppm; SO_2 = 0.5 ppm; RCD = 1.35 mg/m^3). The old AQI value is 3.2 whereas using the revised AQI formula it is 2.4 for the particulate fraction and 0.42 for the gaseous portion. Although the AQI values differ between the two approaches, the conclusion is the same in that the value slightly exceeds the recommended upper limit of 3 and 2 for the old and new AQI formulae, respectively.

If the RCD is lowered to 1.0 mg/m^3 (equivalent to 0.75 mg/m^3 of RCD) the old and new (particulate) AQI values would be 2.5 and 1.85, respectively. Bringing these to common terms by dividing by the acceptable upper limits of 3 and 2 gives values of 0.83 and 0.93, respectively. Both, therefore, meet the acceptable criteria for contaminant levels and therefore ventilation rates.

It is interesting to review the exposure conditions used during the GM and Lovelace diesel toxicity studies. Although the GM group did not measure CO_2, O_2 and SO_2 concentrations, estimates of their level in the inhalation chambers housing the animals can be calculated (Table 152). AQI calculations suggest that both Lovelace exposure conditions exampled here exceed substantially the recommended value using either the old or new formula. The higher of the two GM exposures just exceeds the suggested upper limits and the lower of the RCD exposures is in compliance with the AQI recommendations.

In the Lovelace experiments, evidence was presented which showed that the pulmonary clearance mechanisms were overwhelmed, leading to a much-delayed clearance of particulate, with measurable decreases in lung function suggestive of pulmonary fibrosis.

The GM group reported no measurable decrement in pulmonary function at 0.75 mg/m^3 RCD or lower. Similarly, at 1.5 mg RCD/m^3, the lung clearance mechanisms appear to be able to handle the particulate load, again with no measurable change in pulmonary function. However, the lungs did contain high levels of particulate matter (soot).

TABLE 152

Estimated Inhalation Chamber Environmental Indices

	GM		LOVELACE	
RCD (mg/m^3)	1.50	0.75	7.22	3.59
CO (ppm)	6.60	4.70	29.40	16.60
NO (ppm)	6.90	3.70	9.70	5.60
NO_2 (ppm)	0.50	0.25	0.70	0.40
SO_2 (ppm)	0.95	0.47	7.00*	3.50*
AQI (OLD)	3.9	2.0	19.3	10.6
AQI (OLD)/3	1.3	0.7	6.4	3.5
AQI (NEW)	2.7	1.4	14.4	7.9
AQI (NEW)/2	1.4	0.7	7.2	3.9

(*) Estimated on use of fuel with 0.34% S

Moorman has expressed concern over the lack of follow-up following discontinuation of exposure. NIOSH found pulmonary fibrosis, with a resultant decrease in lung function and diffusing capacity, after returning animals to clean air conditions. Thus, although the GM data may have demonstrated only modest pulmonary involvement at the high particular exposure levels, it is not known whether this loss of function might have increased after discontinuing exposure.

The potential for diesel exhaust to cause an increase in the morbidity and mortality of exposed workers, especially from lung cancer, will only be determined from carefully structured epidemiological studies. Unfortunately, it is in this area that the greatest deficit of knowledge exists. Compounding the problem, and possibly rendering the conducting of meaningful studies almost impossible, is the fact that there are relatively few workers at risk. Thus, even in mines where diesels have been in use for a sufficient period of time (20 - 25 years) to allow the development of neoplastic disease, it will be difficult to obtain non-equivocal statistics on the relationship between pulmonary cancer and diesel exhaust exposure. The smoking habits of the population will further complicate the interpretation of the data.

The most relevant and useful epidemiology report to date was the London Transport Workers Study of diesel bus garage engineers and operators from 1950 - 1974. Although the study had many technical deficiencies, such as the lack of cigarette history and diesel emission levels, it was concluded that the incidence of lung cancer was not significantly greater in the garage workers than in transport workers not exposed to diesel emissions.

Recently, Harris subjected the London Transport data to an alternate statistical evaluation and concluded that the "undetected incidence of lung cancer among diesel bus garage workers was no greater than 160% of the incidence of lung cancer among other unexposed employees".

Further, Harris, using sophisticated and complex statistical computations of the London Transport Workers data, together with data from three short-term bioassays of diesel exhaust extracts, estimated that a 40 -65 year-old male exposed to an average increment of 1 microgram/m^3 of diesel particulate for 20 years would have an upper limit of an increased risk of lung cancer of 1%. Exposure for 30 years to an average increment of 10 mcg/m^3 would result in an upper confidence limit of a 15% increase in lung cancer risk. These data were generated in relation to the increased exposure of the general population to light duty diesel emissions. In comparison, an individual who smokes cigarettes for a comparable period has a proportional increase in lung cancer incidence of 1000 to 2000%.

A comparative calculation for the miner exposed to 1.0 mg/m^3 of diesel particulate for 8 hours a day for 20 years yields an upper limit of increased lung cancer risk of 5 - 6 fold compared to the normal population.

Extreme caution must be used in interpreting this data as several, somewhat doubtful, assumptions are used, not the least questionable being the use of non-human laboratory bioassays to predict the relative carcinogenic potencies of a given dose of diesel exhaust in man.

The Lovelace Inhalation Research Institute graphed the reported annual lung cancer risks for various groups of workers (Figure 131). If a diesel particulate exposure of 1000 mcg/m^3, which is not atypical of the level found in dieselized mines, is fitted to the figure the incidence of lung cancer

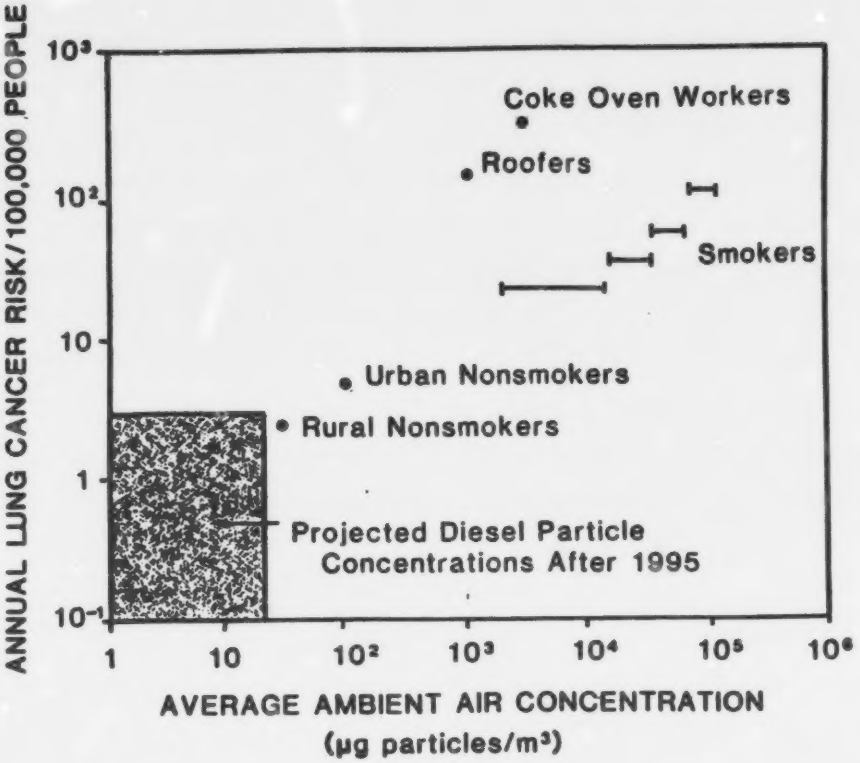

FIGURE 131

Annual Lung Cancer Risk Measured in Coke Oven Workers, Roofers, Smokers and Non-smokers Related to the Concentrations of Particles Inhaled Averaged over all of the Air Breathe During their Lifetimes.

(Source: Reference 486)

in the miner is estimated to be about 110 per 100,000 workers. This compares to 7 and 3 per 100,000 for urban and rural non-smokers, respectively. Smokers (10 -40/day) have a lung cancer incidence of 50 - 80 per 100,000.

These comparison are given, not so much as estimates of risk, but as examples of ways in which maximum risk factors are derived in the absence of epidemiological data. They also demonstrate the difficulty to be expected in trying to measure any significant increase in lung cancer incidence in a population of probably no more than a few thousand individuals.

It must be stressed that there is presently no direct evidence available to demonstrate that exposure to diesel exhaust causes lung cancer in underground mine workers.

There continues to be a paucity of data available relative to the direct effect of diesel emissions on the underground mine workers' health. There is a considerable body of experimental information which strongly implicates the chronic exposure to diesel exhaust as having a potential detrimental effect on pulmonary function and/or in increasing the risk of lung cancer.

Thus, our present attitude is consistent with the recommendation expressed in the 1978 review in that the usual prudent and conservative public health approach be taken to reduce all exposures to as low a level as is practical.

It is the authors' opinion that the AQI formulae, in either of the two forms discussed, can assist in determining acceptable pollutant levels in the dieselized mine and its implementation should provide a reasonable certainty of no significant health effects from the use of heavy duty diesel engines underground.

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